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MAY 1956

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Hyperparathyroidism, with Particular Reference to Treatment *B. Marden Black and James F. Zimmer*

Congenital Vascular Lesions Imitating the Patent Ductus
Carl Davis Jr., Egbert H. Fell, B. M. Gasul, and Robert Dillo

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From MacKenzie et al., p. 824

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A.M.A. Archives OF SURGERY

Doctor of Both Medicines

MICHAEL L. MASON, M.D., Chicago

It is customary for presidential addresses before surgical societies to deal with philosophical aspects of the profession and with the training of the young surgeon. The discourse is usually introduced with some quotation from the writings of the ancients and derives from it certain basic truths as applicable today as they were in ancient times. Not to be outdone by my predecessors, I, too, shall revert to the ancients for inspiration and shall take as my cue the signature of one of our professional ancestors whose true significance in medicine is still a matter of controversy. Theophrastus Bombastus von Hohenheim, otherwise and usually known as Paracelsus, remains today an enigma in scientific history, and the judgment, of the English-speaking peoples at least, has been, in general, unsympathetic to him.

The theme of my discourse is taken from his signature—Beider Artznei Doktor, or, Doctor of Both Medicines. He was among the first in his age to assert that not only medicine but also surgery belonged to the broad profession of healing and that the surgeon as well as the physician should have broad and basic training; both were doctors of medicine.

To us today the necessity of even making such a statement seems absurd; however, in the day of Paracelsus (1493-1541) medicine was the equivalent of internal medicine today, practiced by educated doctors with university degrees, learned in Latin and Greek and in the unchanging and infallible books of the ancients. Surgery, on the other hand, was a low and debased craft; one could hardly call it a profession, carried on by barbers, sheepshearers, hangmen, old women, and itinerant quacks. Paracelsus felt that both branches of the healing art should be united under one appelation of *Doktor* and always insisted upon signing himself Doctor of Both Medicines. Parenthetically, in the writings which stem from his later years, he added to this also Doctor of the Holy Scriptures, but this phase of his activities need not be dilated upon, although his ethical principles for the surgeon were as high as ours of today.

In a way it seems to me we have retrogressed somewhat from the position so vehemently defended by Paracelsus 400 years ago. While he insisted on the union of the two healing arts, we seem to be just as busily breaking up that union into an ever-increasing number of specialties. I do not mean that we should not have specialties; progress in clinical surgery has been due to the intense and concentrated work in restricted fields. No man has the time or ability to encompass all the special fields as an expert. However, there must be some broad, common area of knowledge applicable to both medical disciplines upon which both medicines are based. Paracelsus' plea was that the practice of medicine was based on broad and firm foundations—pillars, he called them—and in the wretched state of knowledge of his day these pillars were largely fanciful, though

Presidential address read at the 63d Annual Meeting of the Western Surgical Association, Seattle, Dec. 2, 1955.

serious, deductions drawn from observation and speculation and based largely on faith. They encompassed the whole field of knowledge at it was understood in his time. They were no better and no worse than the speculations which had gone before and many which appeared after him. It is my feeling that perhaps too little stress is put on this broad common field and too much upon the features which tend to draw medicine apart into specialties. Too early in his career, the student's gaze is directed toward clinical medicine and the specialties. Those interested in internal medicine tend to shun the more practical aspects of the healing art and to develop into medical philosophers. Those whose career is to be surgery tend to neglect the philosophical aspects of medicine and become more and more absorbed in its technical phases.

The general principles of the care of the patient, however, should be the common knowledge of both "medicines." This is basic knowledge, neither medicine nor surgery. The surgeon, because of special training and temperament, is better able to carry out certain technical procedures aimed to "cure" the patient with certain types of disease. The internist, on the other hand, is by training and temperament better fitted to deal with other types of disease. The difference between the two, however, should be mainly in the technical training and not in basic knowledge.

It is my contention that more emphasis should be laid on the early basic training of the student so that when he graduates he is, in fact, doctor of both medicines. Today one would have to say doctor of forty medicines, since specialization has so crept into our profession that possibly even forty might not encompass the field. Internal medicine is not so grievously broken up as is surgery, but we have "heart men" and "stomach men" and "liver men" and "kidney men." New special societies and specialty journals are appearing each year, and one cannot keep up with the new advances made in every field.

It is not my contention that the physician of today should be a specialist in all fields of medicine and surgery or that the internist and the surgeon should be specialists in every area of their respective fields. It is my feeling, however, that the mechanisms of disease as they affect the human body should be so well understood by the physician that it makes no difference what region is affected or what the process might be. Whether he be technically capable of dealing with it is another matter.

Medicine is a learnéd profession whose basis is a general understanding of the structure, functions, and physiologic mechanism of man and a knowledge of the effect upon that mechanism of derangements due to trauma, infection, metabolic and physiologic imbalance, new growths, and disturbances in development. The learnéd physician must have this knowledge and it should be required of him before, not after, specialization.

What we need are learnéd physicians whose knowledge encompasses all the fields of medicine, even though their activities may be restricted to one small field. But, you will say, the field of medicine is too broad for one man to cover adequateiy even in several lifetimes. And I shall have to acknowledge that you are correct if you include in that knowledge the acquirement of the various technical skills which go into the necessary training of our surgical specialists. You will again say that the first two years of medical training consist of lectures, demonstrations and laboratory work dealing with the various sciences upon which medicine is, in fact, based. And that point also I shall also have to give you. Whether two years devoted to these sciences is or is not enough, I am in no position to say. What I do feel, however, is that more and stronger emphasis should be placed on these studies as areas of knowledge in themselves rather than simply as courses to be passed to enable the prospective physician to understand the vocabulary of his chosen profession. The student should not be made to feel that here is simply an obstacle course which he must negotiate successfully if he is to be permitted to emulate

the upper classmen, carry a stethoscope, and discuss "cases."

Obviously, the student cannot make himself as well informed in anatomy as his professors or as skilled in tissue diagnosis as the pathologist. He should, however, derive from these sciences enough of the fundamentals to enable him to understand the disturbed function and deranged anatomy caused by disease.

It is undoubtedly stimulating for the freshmen students to see a patient, let us say, with severe pneumonia, or to witness the removal of an acutely inflamed appendix. We sometimes need glimpses of the goal to cheer us on and give some meaning to our efforts. But this phase of his training should not detract from the more important part of his early schooling: the study of the basic sciences. His business at this time is not clinical medicine; it is anatomy and physiology and the other sciences, and they contain in themselves enough and more of importance to occupy his whole time. These clinical glimpses should be so selected as to illustrate general principles and not specific entities.

The medical soldiers and corpsmen learned a great deal about the practical care of wounds and casualties. Many learned to apply casts and even to give anesthetics and to assist at operations, but to claim that this was medical training would be absurd.

The student has but one time especially set aside, and all too short it seems to me, to learn those things upon which his future practice will be based; he has a lifetime afterward to observe disease. The more factual information he can carry in with him and the better his understanding of the fundamentals, the more valuable will be his observations.

I cannot concede any virtue to the argument that much that is learned in the basic sciences is of no practical value to the busy practitioner. Serious, conscientious study of the sciences is of value in itself regardless of immediate, or even eventual, practical use. But if one still insists upon being practical, who knows how valuable some tiny fact learned today may become when the active mind of an intelligent man makes use of it. The history not only of medicine but also of all human endeavor is full of the practical utilization of scientific curiosities.

Our lack of appreciation of the basic sciences in our medical schools and colleges does not lie in the medical school, it is a more fundamental characteristic of our whole educational system. We have in general a lack of respect for those men whose teachings and discoveries are of the most value to us—namely, our teachers and scientists. The teacher is woefully underpaid and regarded as incompetent to gain a living in any other manner. It is only his inherent sense of duty and high ethical standards which keep him conscientiously at his job. His authority in our schools has been curtailed, and he is made to feel inferior to the youngsters with whose training he is entrusted.

The professor is depicted in our newspaper cartoons and journals as a crackpot, with academic gown and tiny mortarboard cap, gazing dazedly at the practical world about him. Surely this is no man to be entrusted with the solution of weighty problems or be given positions of responsibility.

The scientist of today, while we realize that we need him very much indeed to produce our atomic weapons for us, must be most carefully watched. He is, in the main, too irresponsible to be given positions of trust. He must be subjected to severe investigation and is often placed in the ignominious position of having to defend himself against charges brought by persons unknown to him. His whole career and means of livelihood may be irreparably wrecked by unfounded suspicions openly published.

Learning itself is looked upon as snobbish, unless it can be shown to be of "practical" value. True, not everyone has the capacity for deep intellectual attainments, but I am sure that most of us come far indeed from using our brains to anything like capacity. The teaching of trades and crafts in our schools is theoretically giving many a means of earning a living, but I wager that most of our workers have learned on the job. It would

be much more to the point to concentrate on cultural subjects.

Medical educators today are decrying the fact that too early in his educational career the prospective medical student is devoting his time to so-called premedical studies to the detriment of studies in history and literature, music and art, and other broad cultural phases of our present-day civilization. We find many taking a serious interest in the batting average of a baseball team (that may win the $64,000 question), but how many of us have more than a hazy idea of the events leading up to the Civil War or the successive geologic phases of the earth? Such things we leave to our professors and take little serious intellectual interest in them beyond that needed to pass some required course.

Not until we, as a nation, have developed a greater appreciation of the learnéd man and a respect for learning can we hope to remedy this situation. Success today is measured in dollars, in fine automobiles and fancy apartments, on the billboard, the radio, and the television screen.

We are a people of impatience. This, too, may be a factor. We want to arrive too fast. We are impatient with delay and cannot be bothered with studies whose immediate application is not apparent. We must arrive at once and make our studies count. Unless they can be shown to have practical use, why bother with them? The tendency today, it seems to me, is to stress the application of knowledge at the expense of gaining it.

The surgeon, whether he be interested in orthopedics or ophthalmology, geriatric surgery or gynecology, should have a thorough grounding in those underlying processes which constitute life. He should be a learned man and not simply a skilled technician. Much of the time spent in preparing for a specialty consists in relearning, or possibly even learning for the first time, certain fundamental facts in anatomy and physiology which should have been learned in the first few years in medical school. Practically all our boards require for certification a six months' course in the basic sciences. Granted that these sciences are not static but are constantly changing and require reviewing from time to time, the need of such review is well recognized in medical education. This emphasis, however, should obtain throughout the whole medical curriculum.

This stress upon basic principles as the foundation of medicine could well be carried over into clinical teaching, where the student should be taught broad general principles underlying all of medicine and surgery. It is upon an appreciation of these principles and not by learning by rote that the surgeon can make intelligent decisions. We speak of training in general surgery—perhaps we should do away entirely with the word "general" and speak only of surgery, without the restrictive adjectives. Surgery at once is divorced from regions and systems, it becomes basic and fundamental, a science in and for itself. It is then possible to think in terms of fundamentals, since we are not required to think in terms of any particular surgical act or surgical disease but only in terms of surgical principles. It should make little difference whether we study a carbuncle or a case of appendicitis, a fractured femur or a lacerated forearm, whether we are called to examine a person with obstructive jaundice or renal colic. The surgical management of wounds, for example, is based upon a knowledge of wound healing and upon the factors which favor or retard it. The student who appreciates this process can apply the knowledge to the management of any wound, regardless of its location or nature. Basic operative technique itself has developed from studies on wound healing, on the reaction of tissue to trauma, on the mechanism and physiology of repair and the many factors which influence it. This is basic knowledge indispensable in all of surgery.

I do not suggest at all that the development of specialization be discontinued, but I do feel that the foundations upon which the specialties are built should be broadened and strengthened. Interest in the study of the basic sciences must be developed as a study in and for themselves, not simply as

a stepping stone to future practice. It is only by developing true and intelligent interest that these sciences may become part of the student and his mental processes.

To do this we must begin before the student reaches medical school; we must start even before his collegiate days to inculcate in him a respect for learning and an appreciation of the great cultural activities of the world of literature and music and art, of the historical and economic and social factors in our history. All these things should form the broad base upon which the student finally approaches his study of medicine—not as an isolated subject, without reference to current life but against a well-understood and appreciated background of present day culture.

It seems to me the whole problem is one of attitude on the part of the medical student, and this depends less upon his teachers than upon certain other factors over which the teacher has no control.

If we could develop in the student one-half as much enthusiasm for his course in English literature as there is evidenced in the football team or senior prom, if the same intense desire to understand even imperfectly the general principles of chemistry or the formulae in trigonometry as in figuring out the relative merits of football players, then we would go a long way toward developing a respect and an interest in some of the more important phases of our present-day life.

Now, obviously, it would be a dull world indeed if our daily conversation consisted largely in erudite observations on abstruse problems, but intelligent conversation about topics other than sports and current politics can be interesting and still leave place for less erudite talk. Relaxation we should have, and it should be taken seriously, but it does not necessarily consist in mental blackout.

Most of us, certainly those of us in the medical profession, have been exposed to all the great cultural movements of history; we have seen great pictures, we have heard great music, we have been required to read excerpts, at least, from most of the great books, we have been required to carry out experiments in the various laboratories of chemistry, physiology, and physics. How little, however, do we appreciate these experiences at the time we are having them.

We are urged to extend further and further the training of our young doctors. They are plied with course after course; each professor insists upon special recognition and adequate hours for presentation. It is my impression, also, that we do not pour enough into our students but that what we need is not more time and more courses but more interest on the part of the student. We need a more absorbent sponge. We need a more serious attitude toward learning—not isolated facts, but basic principles. Not until the attitude is changed can we hope to broaden the base of medical education. True, the student must have facts, many facts, but these must be correlated and systematized facts which can only be gained through a grounding in principles. Only in this way can medicine be appreciated as a whole integrated structure rather than a conglomerate of isolated specialties. Upon such a solid foundation the specialties may then build as high as they wish.

The Treatment of Carcinoma of the Hypopharynx and Cervical Esophagus

ORVILLE F. GRIMES, M.D.
and
H. BRODIE STEPHENS, M.D., San Francisco

The proper management of carcinoma of the hypopharynx, larynx, and cervical esophagus presents manifold problems. Threatening as it does the patient's ability to breathe, to swallow, and to speak, the carcinomatous lesion of this area can cause a degree of distress equal to or greater than that of almost any other malignancy. The problems to be dealt with in the care of such patients must be met surgically in the majority of cases. Although the wide resection necessarily includes removal of the larynx, most patients will accept loss of the ability to speak if the procedure provides a chance for reasonably comfortable prolongation of life. In many instances surgical treatment alone affords some measure of relief from the oftentimes intolerable manifestations of the malignant lesion in this area.

Radiation therapy, which is quite successful for malignancies in the region of the oral cavity and nasopharynx, is generally ineffective in the treatment of carcinomas arising in the piriform fossa or the lateral pharyngeal wall as well as those in the postcricoid and cervical esophageal regions. Oftentimes, in these areas the carcinomatous lesion may originally be either overlooked or regarded as a chronic nonspecific ulceration until the subsequent discovery of a cervical lymph node deposit reveals the correct diagnosis. Moreover, malignant lesions in and about the hypopharynx are prone to metastasize to the regional cervical lymph nodes at a rather early stage in their development so that frequently extension beyond the original focus has already occurred at the time of the first clinical examination. Most authorities agree that when a carcinoma has metastasized to the lymph nodes radiation therapy is not as effective as surgical removal of these deposits.

Several other factors also encourage a surgical approach to these lesions. The metastases are often restricted to the local area for prolonged periods. Not infrequently, a lesion arising in the hypopharynx may spill over extensively into the larynx and upper cervical esophagus, yet only minimal and localized lymph-node metastases will be present. It is also true that in spite of a neglected primary lesion the large metastatic deposit which often develops, particularly in the upper cervical lymph-node area, may be the only involvement in the entire lymph-node chain. Because of these factors, an extensive operative procedure even in so-called "late" cases may yield better results than operative treatment of similar lesions lower in the esophagus.

During the past six and one-half years (from March, 1949, to November, 1955), 25 patients with carcinoma of the hypopharynx, larynx, and cervical esophagus have been operated upon at the University of California and San Francisco Hospitals. The operative procedures were patterned after the original work described by Wookey,[5] extended in most cases to include a unilateral or bilateral cervical lymph-node dissection. The first report of this work was made before the American Association for Thoracic

From the Department of Surgery, University of California School of Medicine.

Read at the 63d Annual Meeting of the Western Surgical Association, Seattle, Dec. 1, 1955.

Surgery in 1952.[3] The present review was undertaken in an attempt to evaluate these operative procedures and to determine the effectiveness of various modifications.

Of the 25 patients only 3 were female. The age distribution ranged from 41 to 72 years; the average age at operation was 57. The lesions were epidermoid carcinomas in all except one case, in which a recurrent adenocarcinoma of the thyroid gland had invaded the larynx, trachea, and cervical esophagus, resulting in almost complete stenosis of the latter structure.

Only one patient had sufficient obstruction of his airway to require preliminary tracheotomy. However, almost all the patients were in distress, manifesting varying degrees of dysphagia and pain, and some had marked diminution of their airways.

RESULTS OF OPERATIVE TREATMENT

In general, the over-all results of operative treatment for malignant disease in and about the hypopharynx are not outstanding. On the other hand, in view of the far-advanced stage of the lesion in many of the cases treated surgically the outcome is not particularly discouraging.

In this series there were four operative deaths (Table 1). In two instances the patients died within two weeks after operation as a result of aspiration of blood or salivary and wound secretions into a low-lying trachea which had retracted downward behind the sternum after being transected at the level of the sternal notch. Another patient died from postoperative shock, while the fourth died from repeated hemorrhages secondary to necrosis of the carotid artery. In the last case extensive preoperative x-ray therapy had resulted in impaired vascularity of the skin flaps.

TABLE 1.—*Carcinoma of Hypopharynx; Operative Deaths*

Patient	Sex	Age	Previous Therapy	Postoperative Day	Cause
1	M	54	X-ray	54	1. Hemorrhage 2. Empyema
2	F	68	None	14	1. Hemorrhage 2. Aspiration of vomitus
3	M	65	None	6	Mediastinitis
4	M	41	None	1	Shock

Thirteen other patients died during the period covered by the study (Chart 1). The maximal survival time was 43 months, the shortest was 4 months; the average survival was 21.2 months. In 5 of these 13 patients local recurrences in addition to widespread metastases were noted, while in 8 patients there was no evidence of recurrence or persistence in the local operative area (Table 2).

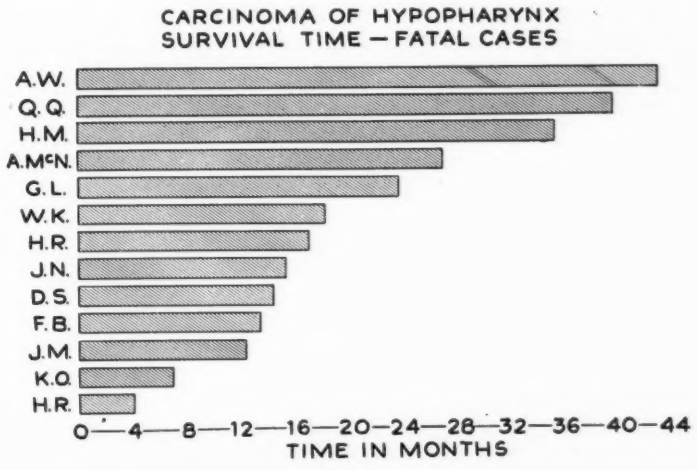


Chart 1.

TABLE 2.—*Carcinoma of the Hypopharynx; Analysis of Fatal Cases*

Cause of Death	
Recurrence or Persistence of Carcinoma	Distant Metastases Without Local Recurrence
5	8

Of the 25 patients in the series, 8 are still alive. Six of these patients are free from recurrent disease (Chart 2). The longest survival in this group is five and one-half years, while the shortest is one year. The other patients in this category have lived in relative comfort for three years or more.

operative therapy extend significantly beyond the relatively small group of patients who survive.

EVALUATION OF OPERABILITY

Evaluation of the extent and operability of carcinoma of the hypopharynx, larynx, and cervical esophagus can be extremely difficult. Frequently, preoperative pharyngoscopy, laryngoscopy, and esophagoscopy provide only incomplete information concerning the extent of the lesion, especially laterally and inferiorly. The criteria of inoperability often do not become evident until the time of operation and, beyond that, frequently only after

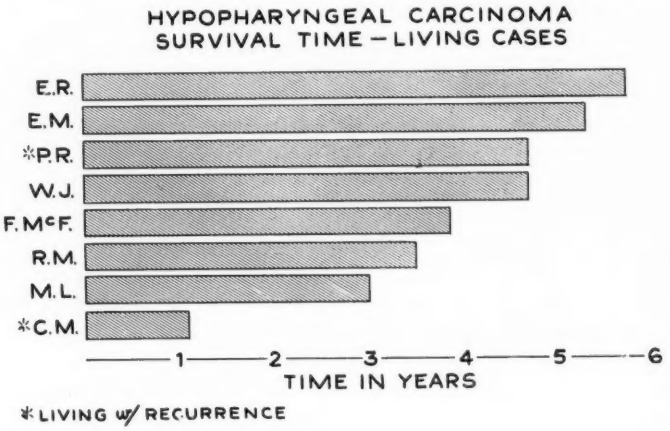


Chart 2.

These figures as well as other statistics in the literature indicate that as yet the chance for a permanent cure is relatively small. However, in this series operative therapy has unquestionably been of marked benefit to those who survived from a few months to several years. Thus, almost all of these patients were relieved of distressing symptoms by the operative procedure. Many of them were subsequently able to resume a fair measure of their normal way of life. A few returned to gainful employment. All of these patients appreciated the palliation afforded them and were grateful for the results achieved in spite of the loss of the ability to speak. Hence, it appears that the benefits of

one has passed the point of no return in the dissection.

Even with the cervical tissues exposed, it is often difficult to determine whether the lesion has extended into areas which cannot be completely removed. On the other hand, extensive masses in these areas may be satisfactorily excised even though they at first appear inoperable. Extension to the base of the tongue or upward along the lateral walls of the hypopharynx does not necessarily indicate inoperability, since these areas can be widely excised and the resultant defects satisfactorily reconstructed. The main difficulty we have encountered is the downward extension of these lesions in the esophagus

and trachea beyond the palpable limits of the tumor. In such cases, almost always the margin of resection inferiorly is narrow and local recurrence is possible. Downward extension occurs especially in the esophagus, where malignant cells may spread through the submucosa for a considerable distance beyond the recognizable margin of the tumor. Gross involvement of the trachea can be managed satisfactorily except in those rare instances where contiguous spread into low-lying regions of the cervical portion of the trachea has occurred.

In this series the local extent of the malignant disease varied considerably. In every case one or more areas in the region of the hypopharynx were involved. Occasionally, one structure in the area, such as the epiglottis, was spared from malignant penetration, and in other cases the lesion appeared to be localized to the piriform sinus and the immediately surrounding area. In no instance, however, did the gross operative findings permit a less radical procedure than en bloc excision of the hypopharynx, larynx, and cervical esophagus.

OPERATIVE PROCEDURES

Laryngectomy was included as a part of the operative procedure in every case. The common lymphatic pool and the close proximity of the larynx to the esophagus and the hypopharynx preclude the possibility of preserving the larynx. None of the patients in this series hesitated to accept laryngectomy along with en bloc excision of the primary lesion. Moreover, the postoperative follow-up has afforded heartening evidence of the patient's ability to adjust either to complete loss of power of speech or to the use of an artificial larynx.

In 17 patients a unilateral radical neck dissection was performed at the time of the definitive operation, either because a large, grossly involved mass was present clinically or because a small, yet suspicion-arousing lymph node was palpable preoperatively. In all but 2 of these 17 cases lymph-node metastases were demonstrated histologically in the specimen removed at operation.

Although it has been reported that the blood supply of the skin flap will be compromised unless the neck dissection is limited and the upper portion of the sternomastoid muscle is preserved,[4] our experience indicates that a standard radical neck dissection can be accomplished without significant hazard in this regard. In a few instances the extreme tip of the skin flap became necrosed, but this tissue loss did not prevent successful formation of a skin-lined swallowing tube in the second stage. Moreover, since most cervical metastases initially appear high in the deep cervical lymph-node chain, their complete removal requires the additional excision of the overlying sternocleidomastoid muscle.

In eight patients unilateral neck dissection was not performed, either because the preoperative evaluation suggested that the patient could not tolerate the extended procedure or because no evidence of metastatic disease was demonstrated by frozen-section diagnosis at the time of operation. Of this group, subsequent lymph-node metastases developed in three patients. On the basis of accumulated clinical experience, even though no grossly palpable lymph nodes exist and even if frozen-section diagnosis is negative for metastatic carcinoma, it is our opinion that a unilateral neck dissection should be performed as part of the primary procedure, provided the patient's condition will permit.

Of those who had radical neck dissection, nodes occurred on the contralateral side in four. Because of the extensive procedure these patients had already undergone, x-ray therapy rather than contralateral radical neck dissection was recommended. However, in view of the imperfect response, we now believe that these patients should have received a neck dissection on the opposite side when palpable nodes developed. In fact, the question arises as to whether an elective neck dissection on the contralateral side should not have been offered shortly after their recovery from the initial operative procedure. Even when lymph-node metastases occur contralaterally, they again tend to remain localized in the cervical region for a prolonged period before dissemination occurs

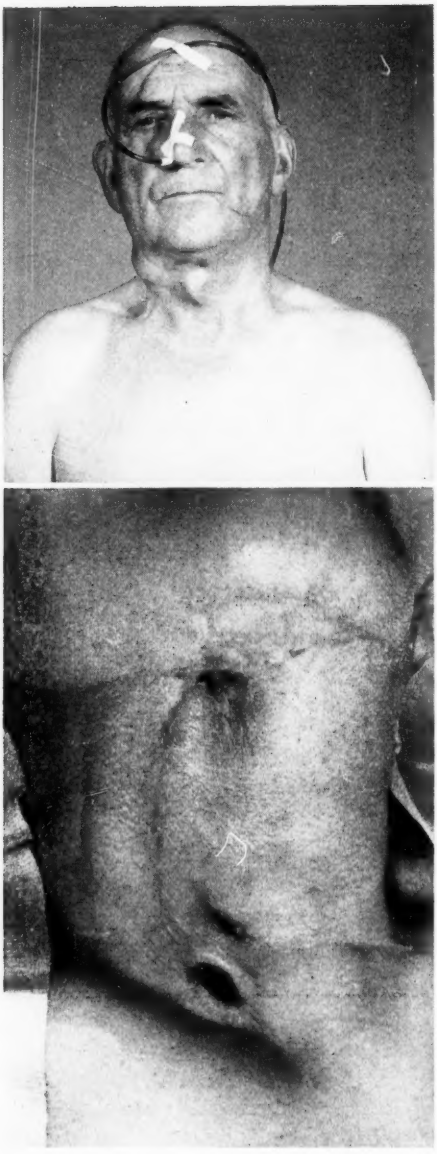


Fig. 1.—*A*, the original lesion was incorrectly diagnosed as a nonspecific inflammatory process of the hypopharynx nine months before a large metastatic lymph node developed on the right side. *B* shows the completed first stage of the operative procedure with the pharyngostome and esophagostome visible at either end of the lateral sinus. A right radical neck dissection was performed. *C* shows the completed skin-lined swallowing tube four years, eight months later. The tube still functions well. Metastases to the contralateral side were treated by radiation.

(Fig. 1). One might logically, therefore, consider a contralateral neck dissection as a prophylactic measure in selected cases.

In 20 patients the multiple-stage resection and reconstruction procedure was employed. The staged procedure consists of excision of all the involved areas, followed by partial reconstruction of the swallowing mechanism by suturing the rectangular flap of skin and subcutaneous tissue circumferentially to the hypopharynx above and the transected esophagus below (Figs. 2 and 3). This first stage permits formation of a so-called "lateral sinus" through which a gastric tube is passed to provide nutrition during the three months which must elapse before the skin-lined tube can be completed (Figs. 4, 5, and 6). During this interval, the blood supply of the flap is insured by development of collateral circulation from the deep tissues of the neck.

In an effort to avoid the prolonged period during which the patient must be fed through an inlying gastric tube, it has been suggested that the entire procedure be performed in one

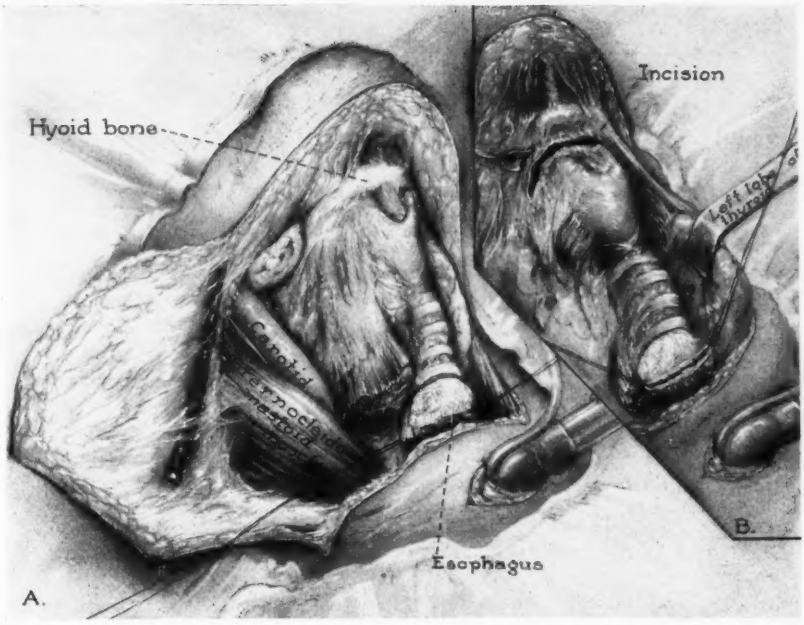
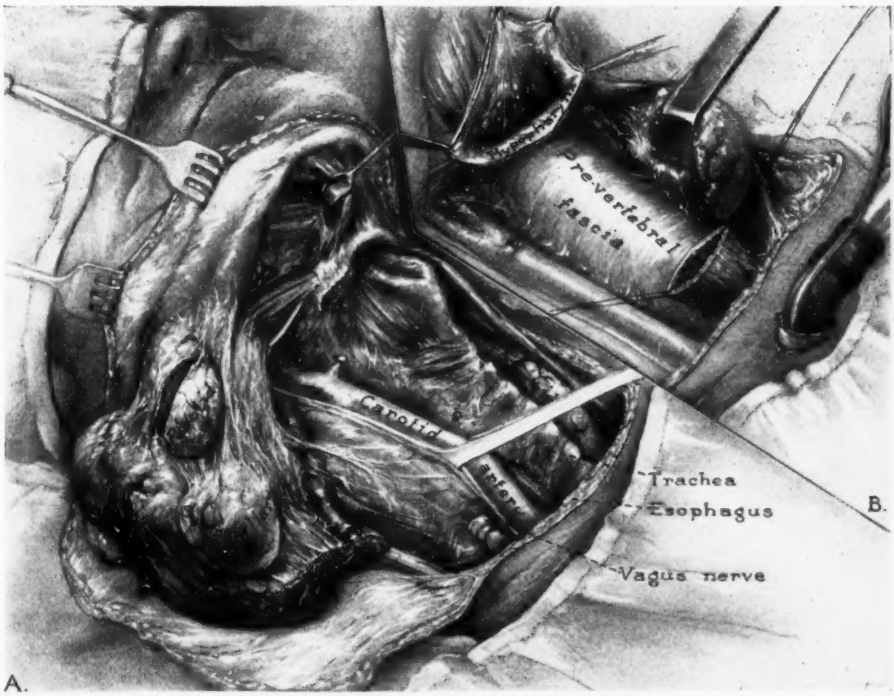


Fig. 2.—*A*, the rectangular skin flap is reflected laterally. The trachea is transexted as high as possible. The tracheostomy is established in the suprasternal notch. *B*, the esophagus is transected well below the tumor. The hyoid bone, although usually spared, can be sacrificed if necessary.

Fig. 3.—*A*, the radical neck dissection is completed before the primary lesion is attacked. *B*, the midline structures are removed on the plane of the prevertebral fascia. A portion of the contralateral thyroid lobe is preserved.



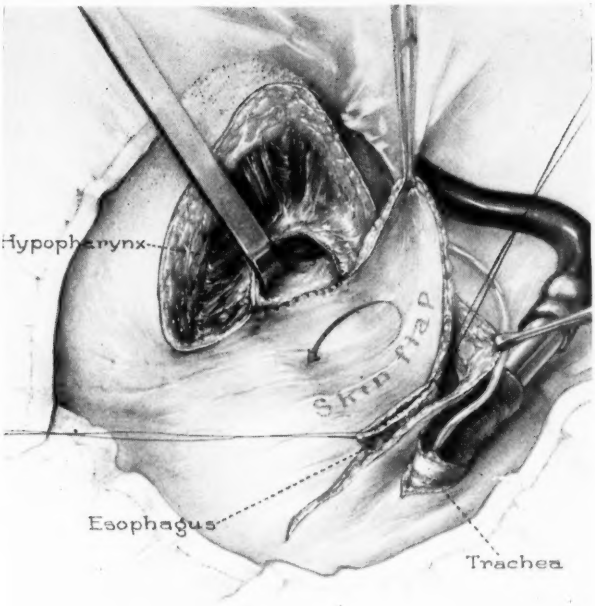


Fig. 4.—The formation of the lateral sinus begins by suturing the skin flap to the posterior hypopharyngeal wall.

Fig. 5.—The circumferential suturing of the skin flap to the hypopharynx and esophagus is completed. A nasogastric tube passes through the lateral sinus into the stomach. The lower portion of the wound is drained.

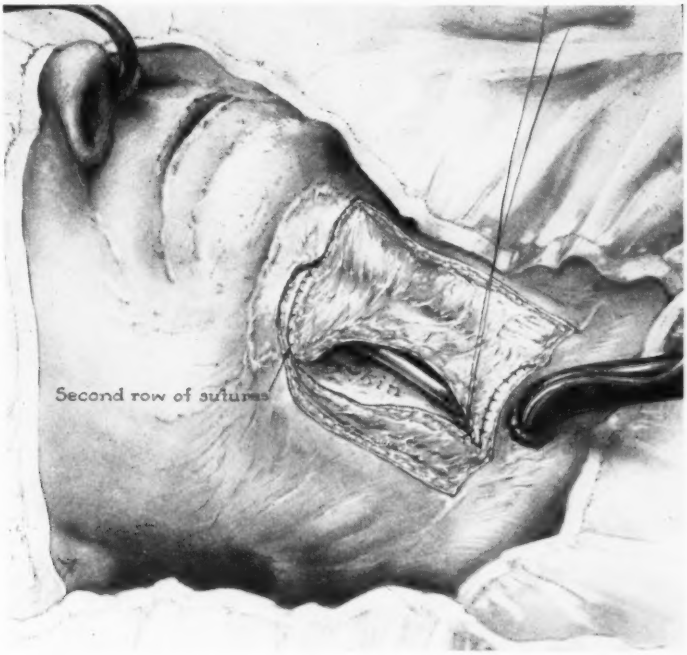


Fig. 6.—Establishment of the skin-lined tube. The freshened edge of the skin flap forming the lateral sinus is sutured to a vertical incision here shown on the right side of the neck. The exposed raw surface is skin-grafted.

stage with reconstruction of the swallowing mechanism by means of a skin graft applied around either a wire mesh or plastic stent.*

In four patients in this series a one-stage procedure was attempted. In two of these patients primary reconstruction was accomplished by means of a skin graft supported by a tantalum-mesh stent. In both cases concomitant radical neck dissection was performed. The initial postoperative course of both patients was satisfactory (Fig. 7). A

* References 1 and 2.

Fig. 7.—Anterior (*A*) and lateral (*B*) views of completed one-stage procedure with a skin graft applied around a tantalum-mesh splint. The initial functional result was satisfactory.

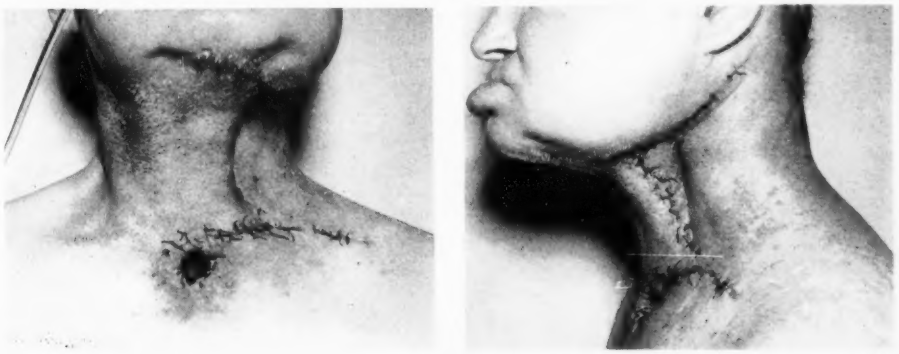


Fig. 8.—Progressive stenosis of artificial esophagus. *A*, the tantalum-mesh splint in place with skin graft wrapped around it. *B*, the size of the skin tube appears adequate shortly after removal of the splint two months after operation. *C*, marked stenosis four months after operation.

salivary fistula developed from the lowermost suture line but healed spontaneously in each instance. Within six weeks after operation, however, both patients complained so bitterly of pain that it was necessary to remove the splint. In subsequent weeks, the skin-grafted tubes in the cervical region became increasingly scarred and stenosed to such an extent that neither patient was able to swallow his salivary secretions (Fig. 8). The scarring and fibrous tissue laid down about the skin grafts were so firm and unyielding that dilatation was impossible. It was therefore necessary to establish a gastrostomy in both patients in order to maintain adequate nutrition.

The scarred cervical skin-grafted swallowing tubes were then reexcised (Fig. 9), and, through a process of skin- and subcutaneous-tissue-flap advancements, new swallowing mechanisms were constructed from tissue obtained from other areas of the body. In

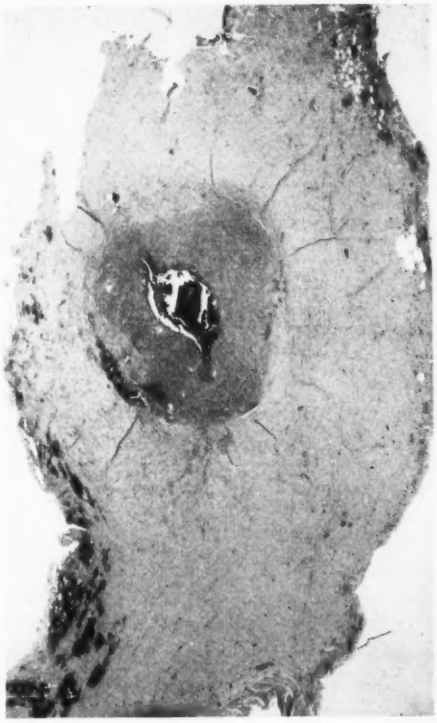


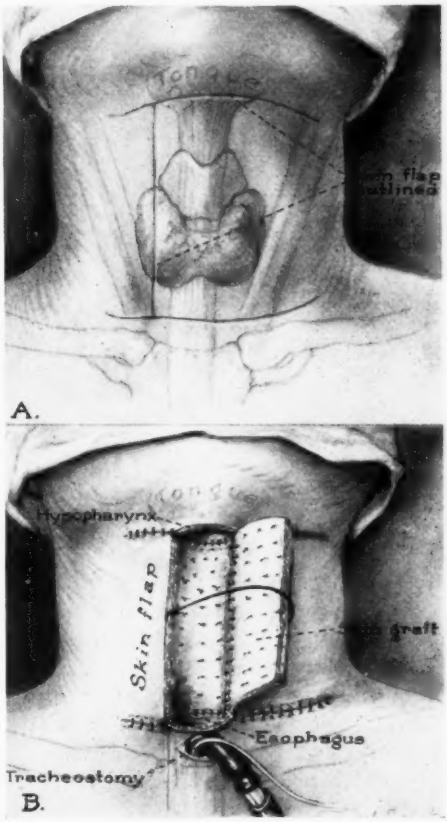
Fig. 9.—Ten-power magnification of the scarred artificial esophagus removed from the patient whose x-rays are shown in Figure 8. Note the marked fibrous reaction around the hyperkeratotic skin graft. The lumen is almost obliterated and is filled with cellular debris.

one case the skin-lined swallowing tube was completed, thereby permitting normal swallowing, only three months before the patient's death from widespread metastases. The other patient, who had had no lymph-node metastases at operation, is living and well, although his swallowing tube is still in the process of completion.

In two other patients an attempt was made to reconstruct the swallowing mechanism by means of a modified one-stage operation at the time of radical resection of the hypopharynx, larynx, and cervical esophagus. The procedure, first attempted in the experimental laboratory, consisted essentially of a skin graft laid on the prevertebral fascia to constitute the posterior wall of the artificial esophagus. The graft was then reflected anteriorly to line the undersurface of the rectangular skin flap (Fig. 10, *A* and *B*), which, in turn, was sutured to the margin of the opposite skin flap, thereby completing the skin-lined tube in one stage (Fig. 11, *A* and *B*).

A review of our own experience and that recorded in the literature suggested that the stent material by its foreign-body reaction in an area bathed with salivary secretions might set the stage for eventual scarring and stenosis reported so frequently as a complication of the usual one-stage procedure. Consequently,

Fig. 10.—*A*, rectangular skin flaps are raised. The longer flap is hinged on the side of the neck dissection. *B*, after a resection of the midline structures, the skin graft is sutured to the subcuticular layer of the right-sided skin flap, to the prevertebral fascia, and to the undersurface of the left-sided skin flap.



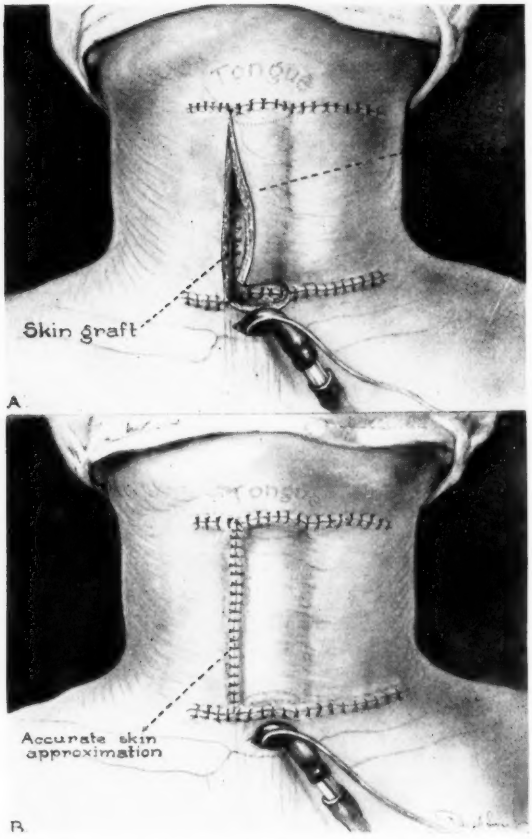


Fig. 11.—*A*, the composite skin flap and graft is circumferentially sutured to the hypopharynx and esophagus. No splint is used. *B*, the actual skin margins of the two flaps are united to complete the swallowing tube.

the skin graft was secured to the undersurface of the skin flap by means of many closely placed sutures in an effort to obtain rapid healing without the use of a splint. In both patients, however, partial sloughing of the graft and some breakdown of the skin suture lines occurred.

We are confident that diminished vascularity of the skin flaps played the major role in the failure of these two procedures. In both patients a radical neck dissection was included in the primary resection because of known metastatic malignancy in the nodal chains. The neck dissection of necessity made the skin flaps long and therefore compromised their blood supply. Since the procedure depends entirely upon the integrity of the extreme distal portion of the skin flap, diminished blood supply to this area was largely responsible for the lack of prompt healing. The use of shorter skin flaps made possible by delaying the necessary neck dissection until a later date may allow early and adequate healing.

When the disease extends below the level of the suprasternal notch, the standard Wookey procedure cannot be accomplished because of inadequate length of esophagus to which a cervical skin flap can be sutured. These advanced lesions frequently invade the

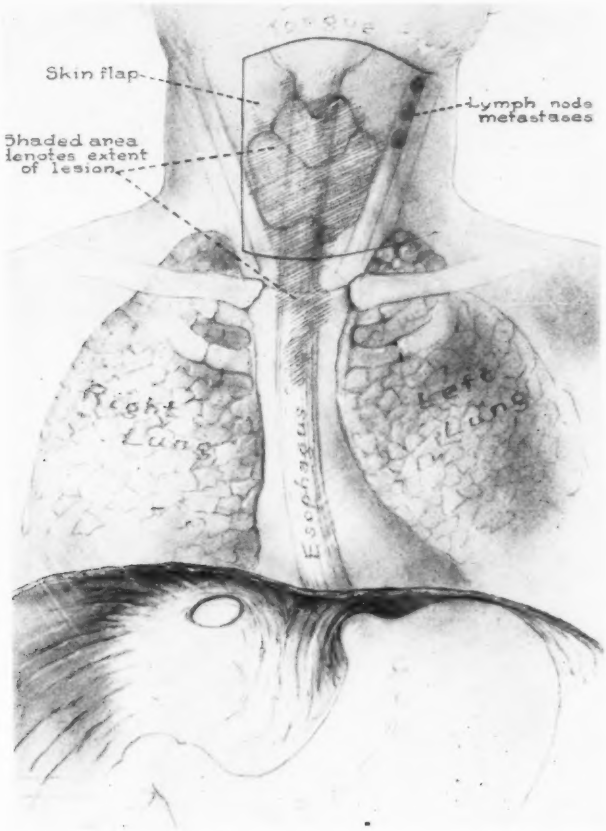


Fig. 12.—Diagrammatic drawing demonstrating the extensive lesion in the esophagus with involvement of the larynx and trachea and extension downward through the superior thoracic strait.

periesophageal tissues laterally and the larynx and trachea anteriorly. Oftentimes, they remain relatively well localized within the midline cervical structures with minimal or no lymph-node involvement, although they may manifest considerable contiguous spread. These extensive carcinomas present difficult problems of treatment.

One patient in our series had a squamous-cell carcinoma in the midline of the neck involving almost the entire cervical esophagus and the larynx, with spread to the periesophageal tissues on both sides. The lesion extended below the level of the clavicles as well (Fig. 12). It was planned to resect the entire esophagus, including the intrathoracic segment, along with the involved portion of the larynx and trachea, and then to advance the stomach through the thorax to a level low in the cervical region where the first stage of a Wookey procedure could be accomplished by suturing the skin flap to the hypopharynx above and to the gastric fundus below. At operation, however, the stomach was found to be long and extremely mobile, allowing the entire cervical region to be spanned by this structure. The fundic portion of the stomach was anastomosed to the hypopharynx posteriorly and to the base of the tongue anteriorly (Fig. 13). Unfortunately, this patient developed a severe and resistant

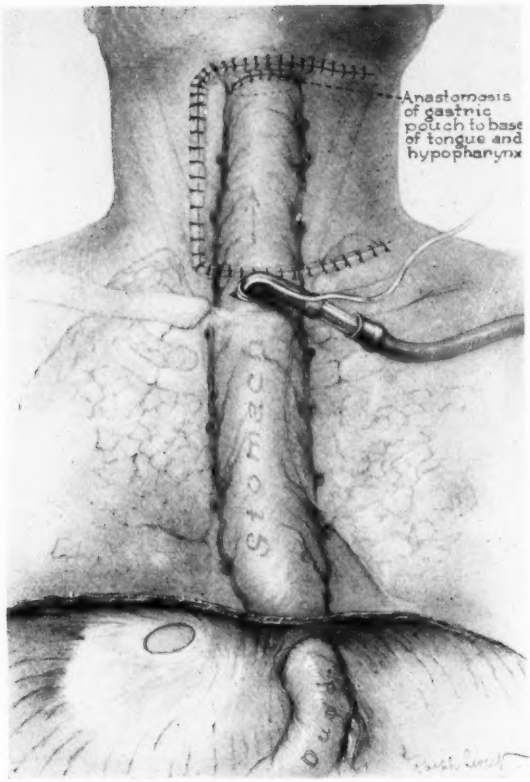


Fig. 13.—The skin flap was raised in anticipation of its union with the transposed stomach at the level of the clavicles. Because of the mobility of the stomach, a primary anastomosis between the stomach and the hypopharynx was possible.

hypotension and died 48 hours after operation. Postmortem examination revealed no evidence of residual carcinoma, and the high-lying anastomosis appeared to be in excellent condition.

Procedures such as this, possibly even divided into stages, may well prove of value in the management of those lesions which extend into the region of the superior thoracic strait from their site of origin higher in the cervical region.

SUMMARY AND CONCLUSIONS

Although the over-all results of surgical treatment of carcinoma of the hypopharynx, larynx, and cervical esophagus are not outstanding, they are not wholly discouraging in view of the far-advanced stage of the lesion in many cases and in view of the substantial benefits derived from operation by those who eventually succumb to the disease. In this series of 25 patients there were four operative deaths. Eight patients are still alive, six without recurrence. The longest survival is five and one-half years. The remaining 13 patients died during the period covered by the study. The survival period in this group ranged from 4 to 43 months. All of these patients were grateful for the palliation afforded by the operative procedure, even though ability to speak was necessarily sacrificed.

Although there are reports in the literature suggesting the use of a one-stage procedure for the surgical treatment of malignant disease of the hypopharynx, larynx, and cervical esophagus, analysis of our series seems to indicate that the staged procedure is satisfactory. The skin-lined swallowing tube produced in this manner is soft, pliable, and functional, in contrast to the firm, indurated, narrow artificial esophagus made by means of the various skin-grafting procedures. The period of delay between stages appears to be a small price to pay for a lasting physiological swallowing apparatus.

REFERENCES

1. Conley, J. J.: A One-Stage Operation for the Immediate Reconstruction of the Pharynx and Cervical Esophagus Following Radical Resection, West. J. Surg. **63**:344-347 (June) 1955.

2. Edgerton, M. T.: One-Stage Reconstruction of the Cervical Esophagus or Trachea, Surgery **31**:239-250, 1952.

3. Grimes, O. F., and Stephens, H. B.: The Surgical Treatment of Carcinoma of the Hypopharynx, Larynx, and Upper Esophagus, J. Thoracic Surg. **24**:246-255 (Sept.) 1952.

4. Mustard, R. A.: Reconstruction of the Oesophagus, S. Clin. North America **34**:979-995 (Aug.) 1954.

5. Wookey, H.: The Surgical Treatment of Carcinoma of the Pharynx and Upper Esophagus, Surg. Gynec. & Obst. **75**:499-506 (Oct.) 1942.

Cancer of the Esophagus

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Carcinoma of the esophagus was considered beyond the scope of surgical cure until 1913, when Franz Torek [1] reported the first successful esophagectomy with anterior esophagostomy for malignancy of the thoracic esophagus. It was not until 1938 that Adams and Phemister [2] reestablished the continuity of the gastrointestinal tract following an esophagectomy by performing a transthoracic esophagogastrostomy. The glow of enthusiasm following these technical advances has diminished somewhat in recent years in the light of impartial analysis of survival statistics, and today there is outright skepticism from many quarters as to the plausibility of surgical cure of carcinonma of the esophagus. The few patients who are alive and well several years after their expected deaths and the larger number who have been made more comfortable by the procedure provide sufficient stimulus for some surgeons to continue recommending the surgical approach. The greater number of patients who have not survived a significant period of time following their operation afford ample reason for other surgeons to recommend abandoning an arduous procedure with so little statistical advantage. A stronger response has been felt toward the former attitude by the staff of the surgery department, Wayne University College of Medicine, at the Detroit Receiving and Dearborn Veterans Hospitals.

The present study is a review of 124 cases of primary carcinoma of the esophagus initially treated at the Detroit Receiving and the Dearborn Veterans Hospitals during the eight-year period from January, 1947, through December, 1954. Neoplasms invading the esophagus from contiguous structures, particularly the stomach, were not included in the survey. There were 115 men and 9 women (Fig. 1). The pronounced majority of men in this series reflects the preponderance of males in the population of the Veterans Hospital. There were 80 white and 44 Negro patients, with the highest incidence occurring in the seventh decade. The youngest patient was 28 years and the oldest 89 years, with an average age of 57 years.

Dysphagia was the presenting symptom in 90% of the patients (Fig. 2). Two patients presented themselves to their physicians with the complaint of cervical metastases, and three presented themselves with anorexia and weakness. Six patients were admitted to the hospital complaining of epigastric pain. One patient was hospitalized and was being treated for thrombophlebitis of the lower extremity when a sudden massive hematemesis led to the diagnosis of esophageal carcinoma. In the two patients who presented themselves with cervical metastases, it was 7 and 13 months later before they complained of dysphagia.

In 81 patients the weight loss was definitely known. Seventy-six per cent lost 20 lb. or more and sixteen per cent 50 lb. or more (Fig. 2).

Unfortunately, the cardinal symptom of esophageal malignancy, that of dysphagia, usually occurs only after a silent period of growth during which the esophagus is being converted into an inelastic rigid tube by complete encirclement of the malignant lesion. Because the esophagus lacks a confining serosa and is so closely approximated to other vital structures, the carcinoma is frequently incurable when this stage is reached.

Not only is the ability of the physician to diagnose cancer of the esophagus in an early stage hampered by this silent period of

Read at the 63d Annual Meeting of the Western Surgical Association, Seattle, Dec. 1, 1955.

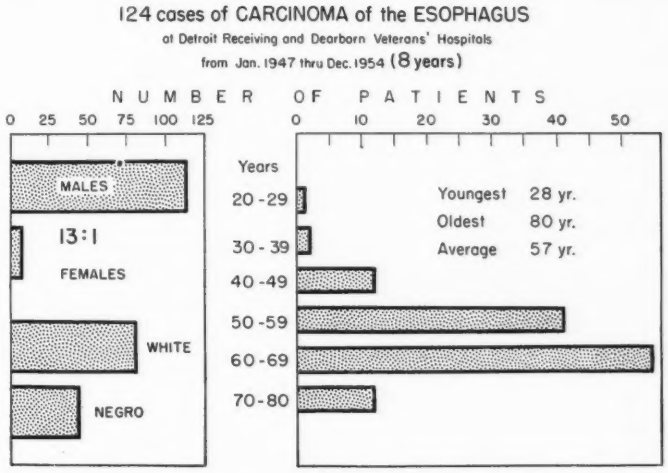

Figure 1

growth, but there is frequently a significant delay between the onset of a patient's symptoms and his contacting a physician. Seventy per cent of the patients in this series experienced symptoms for three months or more and thirty-seven per cent six months or more before admission, with the distribution as shown in Figure 3.

Of primary importance in establishing an early diagnosis of esophageal carcinoma is the making of an esophogagram and performance of esophagoscopy on all patients with suspicion-arousing symptoms, such as difficulty in swallowing or substernal distress. Esophagoscopy was attempted in 113 cases in the present series (Fig. 4). A positive biopsy was obtained in 103 instances (83%), and, although the procedure was successful, a positive biopsy was not obtained in five cases. The procedure was unsuccessful in five patients. In 11 cases, none of whom were subjected to operation, esophagoscopy was not performed, and the diagnosis was established at autopsy.

Occasionally there is uncertainty as to whether the carcinoma originated in the lower portion of the esophagus or in the upper

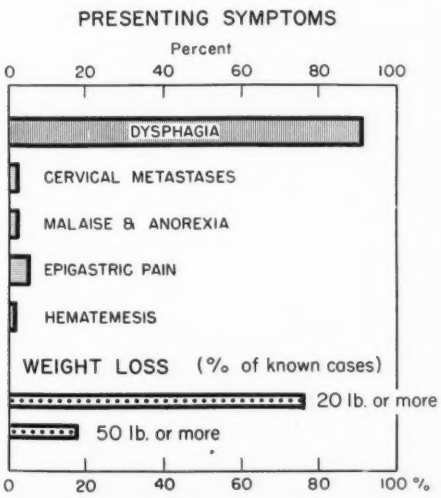

Figure 2

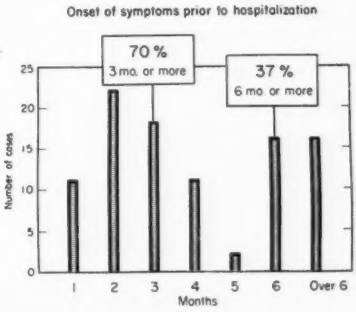

Figure 3

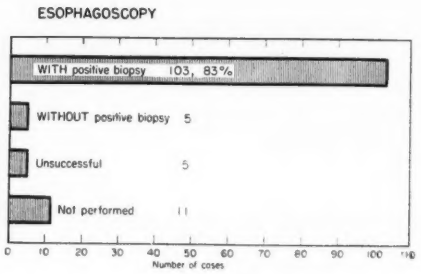
ESOPHAGOSCOPY
WITH positive biopsy 103, 83%
WITHOUT positive biopsy 5
Unsuccessful 5
Not performed 11
0 10 20 30 40 50 60 70 80 90 100 110
Number of cases

Figure 4

end of the stomach. Biopsy with histological confirmation will usually settle this point in that squamous-cell carcinoma does not arise from gastric mucosa. Adenocarcinoma, on the other hand, rarely arises from esophageal mucosa.[3] The histological diagnosis in 94% of the patients in this survey was squamous-cell carcinoma (Fig. 5). There were four cases of adenocarcinoma, two undifferentiated carcinomas, and one adenoacanthoma. The adenocarcinomas, as far as we could determine, were primary in the esophagus.

For purposes of anatomical classification the esophagus was divided into four segments: cervical, upper thoracic, midthoracic, and lower thoracic. The cervical segment is that portion extending from the cricopharyngeus muscle to the upper border of the clavicle; the upper thoracic segment extends from the clavicle to the superior portion of the arch of the aorta; the middle segment includes that part behind and below the aortic arch for a distance of approximately 4 cm., and the lower segment is the remaining

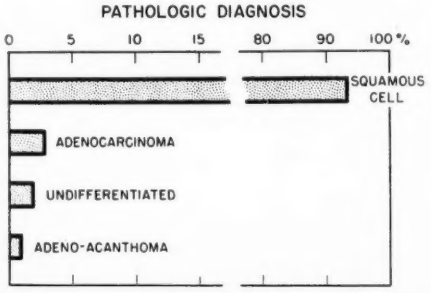

Figure 5

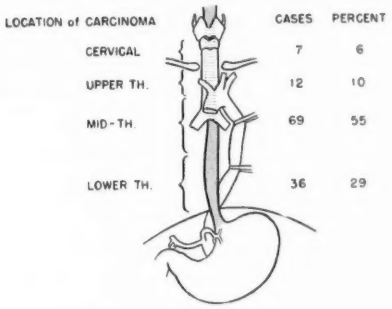

Figure 6

esophagus (Fig. 6). In the operative cases the location of the lesion was determined by the surgeon's operative report. In the non-explored cases the findings at esophagoscopy and the esophagogram were used to determine the location of the lesion. There were 7 cases (6%) located in the cervical esophagus, 12 (10%) in the upper thoracic, 69 (55%) in the midthoracic, and 36 (29%) in the lower thoracic segment.

Of the 124 cases 83, or 67%, were operable and 41, or 33%, were nonoperable (Fig. 7). Forty-two per cent of the lesions in the operable group were resected. The operating surgeon believed there was a chance for cure in 20%, and in 22% a palliative resection was performed. The lesion was not resectable in 25% of the patients.

The reasons for nonresectability in the operated group were as follows: local extension in 29 cases, distant metastasis in one, and poor condition of the patient in another. In those with local extension there was obvious involvement of the immediately ad-

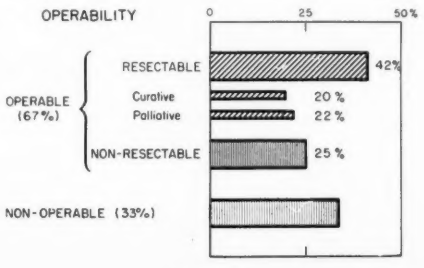

Figure 7

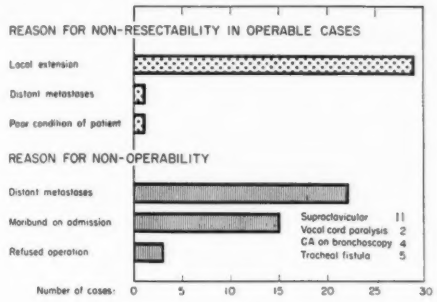

Figure 8

jacent vital structures, such as the aorta or bronchus (Fig. 8).

Thirty patients were considered inoperable. Twenty-two of these had supraclavicular node metastases or other manifestations of advanced disease, such as vocal cord paralysis or extension of the carcinoma into the bronchus or trachea. Fifteen patients were moribund on admission and three refused surgery (Fig. 8).

A death was considered an operative mortality if the patient died from any cause within 30 days of the esophagectomy or if a complication of the operation ultimately led to his death, regardless of the postoperative period. Of the 52 esophagectomies there were 25 operative deaths, or an over-all operative mortality of 48%. There was a 29% operative mortality in the curative and 64% in the palliative group (Fig. 9).

There were three resections of carcinomas involving the cervical portion of the esophagus with an operative mortality of 33% (Fig. 10). A local resection popularized by Wookey [4] was performed in each of these cases.

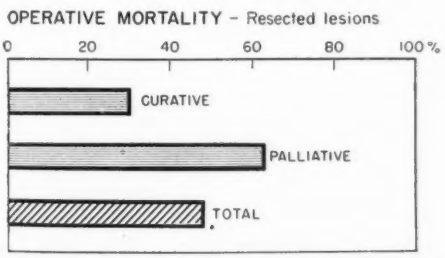

Figure 9

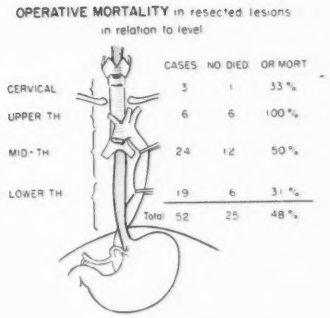
OPERATIVE MORTALITY in resected lesions in relation to level
CASES NO DIED OR MORT
CERVICAL 3 1 33%
UPPER TH 6 6 100%
MID-TH 24 12 50%
LOWER TH 19 6 31%
Total 52 25 48%

Figure 10

There were six resections for carcinoma involving the upper thoracic segment, with a 100% mortality. In these instances an esophagogastric anastomosis was performed in the neck through a separate incision anterior to the sternocleidomastoid muscle after the initial esophageal approach had been carried out transthoracically. There were 24 resections for lesions of the midthoracic segment, with an operative mortality of 50%. In these instances the anastomosis was made above the level of the aortic arch. There were 19 resections for lesions in the lower esophagus, with an operative mortality of 31%. In this latter group the anastomosis was accomplished below the arch of the aorta(Fig. 10).

There were 11 operative deaths due to faulty technique, 5 occurring in the curative group and 6 in the palliative. Two deaths were attributable to gastric infarction, eight patients died as a result of anastomatic leaks, and one died secondary to intra-abdominal hemorrhage. Fourteen deaths were of a nontechnical nature, two occurring in the curative group and twelve in the palliative. Ten

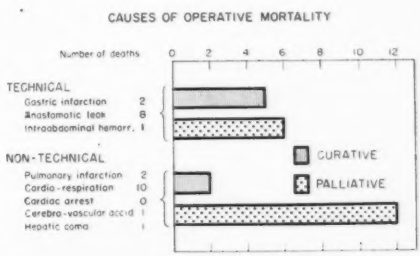

Figure 11

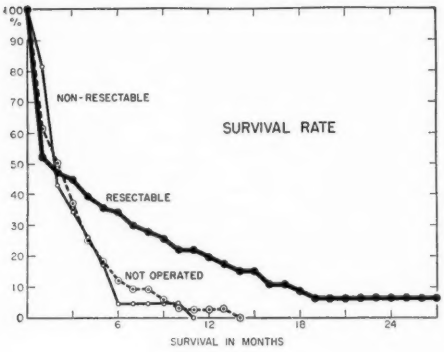

Figure 12

patients died of cardiorespiratory difficulties, two of pulmonary infarction, one of a cerebral vascular accident, and one of hepatic coma. The fact that 12 of the 18 deaths in the palliative group were of a nontechnical nature would suggest the poorer general condition of the patients in the group.

The duration of life in the nonoperated and operated groups, with the latter divided into resectable and nonresectable, is graphically shown (Fig. 12). The abscissa is the length of survival in months after operation, or after diagnosis in the nonoperative cases, and the ordinate is the percentage of living patients. In general the graph demonstrates that those patients in whom the lesion was resected live longer if they survive the operation. The immediate precipitous drop in survival in the resected group reflects the operative mortality. All the nonoperated cases were dead at the end of 14 months and all the nonresected cases at the end of 12 months. Those that survive the immediate operative mortality in the resected group are relieved of dysphagia and for a time enjoy a relatively normal existence. In the nonoperated and in the nonresected groups life from the time of diagnosis until death was miserable. Palliative procedures, such as gastrostomy and jejunostomy, do not relieve suffering in these patients.

There are no patients in this series living more than five years. There are three patients living and well without evidence of recurrence four years after their operative procedure, resulting in an absolute four-year survivorship of 2.4% (Fig. 13). The four-year survival rate in those resected for cure is 12.5%.

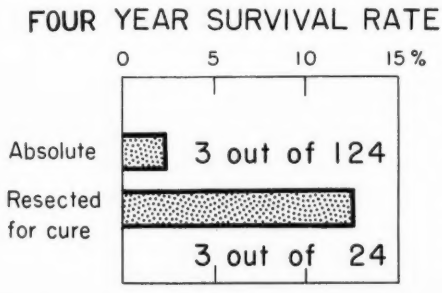

Figure 13

COMMENT

Like carcinoma of other regions, esophageal carcinoma must be discovered in an earlier stage before a significant improvement can be expected in the number cured. This involves education of the public as well as of the physician in respect to the early symptoms of esophageal cancer and the necessity of following through with diagnostic procedures to exclude its possibility. As mentioned by Adams,[9] "the wave of pessimism regarding surgical treatment of this malignant tumor may delay this educational process for some time to come; and surgeons who have become discouraged are inclined to perform operations for the alleviation of symptoms rather than for removal of the neoplasm."

It should be emphasized that the primary objective of cancer surgery is complete eradication of tumor tissue, with resultant cure. At the present time en bloc resection of the malignancy with draining lymph nodes and intervening tissue is the only method of manifesting a cure in cancer involving deep-seated organs, such as the esophagus. Barring any of the contraindications to exploration mentioned above, it is our belief that a transthoracic exploration of an esophageal cancer should be performed in an effort to determine the feasability of cure. In every instance where there is a possibility of eradicating all grossly carcinomatous tissue, esophagectomy

with reestablishment of the gastrointestinal continuity should be performed.

In those cases where the lesion at operation is found to be incurable because of spread beyond the limit of resection, excision of the obstructing malignancy with reestablishment of gastrointestinal continuity can be considered. Certainly in lesions of the lower esophagus this is the procedure of choice.

For those unfortunate persons who present themselves to the physician with obvious distant metastases, palliative procedures to make the patient comfortable for his remaining days, such as gastrostomy, jejunostomy, x-ray therapy with or without the passage of a Levin tube, is certainly indicated.

The operative mortality of esophagectomies in various reported series ranges from 12% to 50%.* Our over-all operative mortality of 48% is high owing to our philosophy of performing palliative resections for the relief of obstruction, to the inclusion in our study of patients with malignancy involving the upper third of the esophagus, where the mortality is high, and to the exclusion of patients with gastric carcinoma, where the mortality is low.

Operability, resectability, and survivorship vary with the selection of patients and will be higher in instances where the surgeon comes in contact with more favorable cases. In the present series all patients with carcinoma of the esophagus were included in the survey, regardless of the stage of the disease. Indeed, a number of patients died before a positive diagnosis could be made, and in two instances death occurred in transit to the ward from the admitting room. The true nature of the ailment in these latter instances was ascertained at the autopsy table. The prospect of three patients surviving four years, of the total of 124 patients admitted, presents a very dismal outlook. However, only 24 of the patients were treated in a favorable stage, when the surgeon was of the opinion that a cure might be manifested by wide resection. Three patients living and well after four years, of the twenty-four resected for cure, is not nearly as hopeless a situation as the figures at first glance might indicate.

* References 5, 7, 9-11.

SUMMARY

Dysphagia, the cardinal symptom of esophageal cancer, unfortunately, means that the disease is advanced, and as a result the number of patients who can be resected for cure is small (20% in this series).

Our operability and resectability rates of 67% and 42%, respectively, are high in comparison to other series.

Our over-all operative mortality of 48% is high owing to (*a*) our philosophy that resection for palliation is justified, (*b*) the inclusion in our statistics of carcinoma of the upper third of the thoracic esophagus, and (*c*) the exclusion of carcinoma of the stomach with secondary extension into the esophagus.

Three patients are living and well more than three years after operation without evidence of recurrence.

Patients who withstand an esophageal resection enjoy a longer duration of life than those in the nonoperative group and live their remaining days much more comfortably.

We believe that a carcinoma of the esophagus, regardless of location, should be resected if there is a possibility of completely removing tumor tissue. A palliative resection is certainly justified if the lesion is located in the lower third of the esophagus, where the anastomosis can be made below the level of the aortic arch, but one must be prepared to accept a higher mortality if a palliative resection is performed for carcinoma of the mid or upper thoracic esophagus, requiring a supra-aortic anastomosis.

REFERENCES

1. Torek, F.: The First Successful Case of Resection of the Thoracic Portion of the Esophagus for Carcinoma, Surg. Gynec. & Obst. **16**:614, 1913.

2. Adams, W. E., and Phemister, D. B.: Carcinoma of the Lower Thoracic Esophagus: Report of a Successful Resection and Esophogogastrostomy, J. Thoracic Surg. **7**:621 (Aug.) 1938.

3. Thomas, J. V., and Hay, J. L.: Adenocarcinoma of the Esophagus: Report of a Case of Glandular Metaplasia of the Esophageal Mucosa, Surgery **35**:635 (April) 1954.

4. Wookey, H.: The Surgical Treatment of Carcinoma of the Pharynx and Upper Esophagus, Surg. Gynec. & Obst. **75**:499, (Oct.) 1942.

5. Parker, E. F.; Hanna, C. B., and Postlethwait, R. W.: Carcinoma of the Esophagus, Ann. Surg. **135**:697 (May) 1952.

6. Wu, Y. K., and Loucks, H. H.: Carcinoma of the Esophagus or Cardia of the Stomach: Analysis of 172 Cases with 81 Resections, Ann. Surg. **134**:946 (Dec.) 1951.

7. Humphreys, G. H., II., and Moore, R. L.: Carcinoma Involving the Esophagus, S. Clin. North America **33**:389 (April) 1953.

8. Garlock, J. H., and Klein, S. H.: The Surgical Treatment of Carcinoma of the Esophagus and Cardia, Ann. Surg. **139**:19 (Jan.) 1954.

9. Adams, W. E.: The Future Outlook of Surgical Therapy for Carcinoma of the Esophagus, Surg. Gynec. & Obst. **100**:366 (March) 1955.

10. Sweet, R. H.: Late Results of Surgical Treatment of Carcinoma of the Esophagus, J. A. M. A. **155**:422 (May 29) 1954.

11. Sweet, R. H.: The Results of Radical Surgical Extirpation in the Treatment of Carcinoma of the Esophagus and Cardia, With 5 Year Survival Statistics, Surg. Gynec. & Obst. **94**:46 (Jan.) 1952.

DISCUSSION

On papers by Drs. Grimes and Wilson, Powers, and Johnston

Dr. Krilik, Seattle: Carcinoma of the esophagus is, comparatively speaking, one of the more unfavorable cancers to manage because of the problems of diagnosis, the biology of the neoplasm itself, and the relative inaccessibility of the lesion to direct treatment. These problems provide innumerable approaches for discussion. However, this time is limited, and only two questions will be briefly reviewed. With full consideration of our present-day methods of treatment and the nature of the disease, how much can we expect to accomplish, and what are our chances for this accomplishment?

In 1952, Drs. Merendino and Mark published a paper in which they contrasted the actual curability and the theoretical curability. They found, on reviewing 100 cases of squamous-cell carcinomas of the esophagus, that 34% of the lesions were theoretically curable—that is, the malignancy was confined to the esophagus or regional nodes. But, on applying such factors as life expectancy of aged patients, operative rejections, refusals of surgery, operative mortality, etc., the actual cure rate would be much below this.

Now, what are the chances of improving the survival rate—earlier diagnosis? "Earlier diagnosis" is a term seen in nearly every published article on cancer. Early diagnosis can also be classified as theoretical and actual. The surgeon is often placed on the spot by a patient or a referring physician on being presented with a so-called early lesion. In conformance with present-day public education on the subject of early diagnosis, he is expected to produce "cures" in these cases. Yet, in reviewing the entire problem, it may be that the longest survivals will be obtained in the patients with long-term histories, owing to the inherent slow anabolic activity of the tumor itself. It is this group that would probably provide the basis for increased survival rate if only some method were available to provide for a diagnosis long before signs and symptoms occur.

Dr. Grimes' presentation was very intriguing and stimulating. He has used the material well, and, if one considers the condition and the age of the patient, the extent of the operative procedure, and the nature of the disease, the operative mortality of 4 of 25 patients is remarkable. His end-results are far superior to the average. This reflects the exacting care that must have been given to all aspects of the problem.

The salient point of his presentation is the recommendation of a two-stage procedure in place of the one-stage reconstruction of the swallowing mechanism. It is agreed that a patent, pliable communication between the hypopharynx and the distal esophagus is essential. The poor results obtained when a skin graft supported by a splint is used is not surprising. Any type of splint is an irritating foreign body. The end-result of irritation is scar. This is well illustrated by the end-results obtained when various prostheses are used in the reconstruction of portions of the trachea.

The case presented, in which the stomach was anastomosed to the hypopharynx and the tongue, is interesting. However, it is felt that if a series of such procedures were done the end-results would be poor. The distant transplantation of the stomach would jeopardize the blood supply at the anastomatic site. One must add to this the constant strain of motion within the hypopharynx and the irritating qualities of the constant flow of saliva.

Dr. Wilson stated that their operative mortality of 48% was high. However, on review of local series and some published series this is within bounds. He has been liberal in his qualification of operative mortality. There is one problem which I believe is very important to the patient and I wish that Dr. Wilson or Dr. Grimes would discuss it. The problem is the indications and contraindications for doing palliative procedures, such as gastrostomies and jejunostomies, in the terminal patients.

DR. ORVILLE F. GRIMES, San Francisco: One of our main difficulties in the surgical treatment of lesions in and about the hypopharynx has been the assessment of operability. Oftentimes these lesions present as an extensive mass in the midline of the neck, extending to both sides and considerably inferior as well. Many times the exact question of whether the lesion is operable cannot be determined until such time as the patient is operated upon, and, beyond that, oftentimes the true assessment of operability is not made until a point of no return has been reached in the dissection.

The tumors which arise in this particular area are prone to metastasize early to the cervical lymph nodes, and lesions arising in the hypopharyngeal area may spill over extensively into the larynx and upper esophagus and yet remain localized for a considerable length of time. It is because of these reasons that an operative procedure in this area, even in late cases, gives results as good as or better than treatment of similar lesions lower down in the thoracic esophagus.

DR. GERALD S. WILSON, Detroit: I should like to thank Dr. Krilik and Dr. Vaughn for discussing this paper. I am thankful to Dr. Vaughn for bringing a little humor into a rather morbid subject.

I am not sure of the place of gastrostomy and jejunostomy for palliation in obstructing cancers of the esophagus. Certainly such procedures do not prolong life, but they may provide some comfort for the patient's remaining days. The best palliative procedure we have devised for those whose lesions are inoperable is passage of a small gastric tube for feeding purposes followed by intensive x-ray therapy. There are some in Detroit who believe that this is the treatment of choice for all cases of carcinoma of the esophagus.

Gastric Volvulus as a Cause of Obstruction

Report of Seven Cases

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RAYMOND W. HAMMER, M.D., Denver
and
WARD C. FENTON, M.D., Rocky Ford, Colo.

Reports in the literature indicate that obstructing gastric volvulus is an infrequent abdominal emergency. Early diagnosis is difficult. Both clinical and roentgenologic evidence is necessary for proper diagnosis. We are presenting the report of seven cases recently encountered for the purpose of reviewing the clinical aspects of the entity, establishing gastric volvulus as a potential postoperative complication, showing that it may be caused by trauma, and further emphasizing the importance of early diagnosis and definitive treatment.

REPORT OF CASES

CASE 1.—A 64-year-old woman had had typical episodes of gallstone colic for 10 years. On the day she was admitted to the hospital, the symptoms became acute and persistent. She had fever, leucocytosis, a rapid pulse, and localized tenderness in the upper part of the abdomen. Exploration the next day revealed an acutely inflamed gall bladder. It was removed, and the abdomen was drained and closed. Her postoperative course was without incident for three weeks. Then she suddenly developed severe epigastric pain and vomited once. The pain was not relieved and it became unbearable. The patient retched constantly, but she was unable to vomit again. Efforts to pass a stomach tube were unsuccessful.

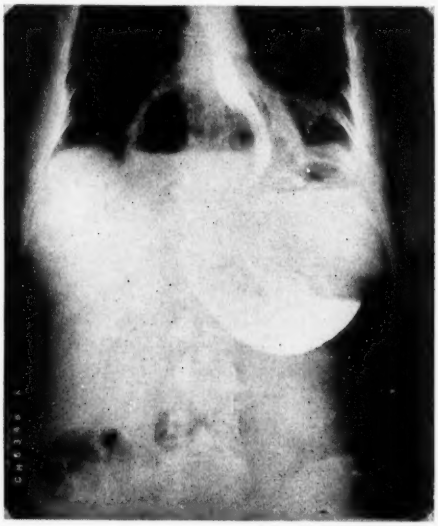
Barium studies (Fig. 1) showed an acute, obstructing organoaxial volvulus associated with a large esophageal hiatal hernia. The greater curvature of the stomach remaining in the abdomen was beneath the left hemidiaphragm. The pylorus was to the left of the midline (posterior to the stomach).

An emergency operation was performed. Most of the stomach was in the left hemithorax. The enlarged esophageal hiatus was thick, and much of the greater omentum was adhered to it. The hernia and the volvulus were reduced from the abdomen, the edematous stomach was resected, and the diaphragmatic crura approximated with nonabsorbable surgical (silk) suture material.

The patient had a smooth convalescence and has remained well.

CASE 2.—A 71-year-old woman had complained of epigastric pain and indigestion for 20 years. An x-ray series of the gall bladder revealed gallstones. A cholecystectomy was performed, and the abdomen was drained and closed. After seven days of uneventful progress, she developed intermittent abdominal pain and vomiting and her abdomen became distended. A plain plate of the abdomen showed an intestinal pattern typical of acute intestinal obstruction. At laparotomy the same day, an obstructing band was divided and the patient's abdomen was closed. She convalesced without incident and returned to her home.

Fig. 1.—Acute organoaxial volvulus associated with large esophageal hiatal hernia. The greater curvature lies beneath the left hemidiaphragm with the pylorus lying to the left of the midline (posterior to the stomach). The stomach is completely obstructed.



Presented at the 63d Annual Meeting of the Western Surgical Association, Seattle, Dec. 1, 1955.

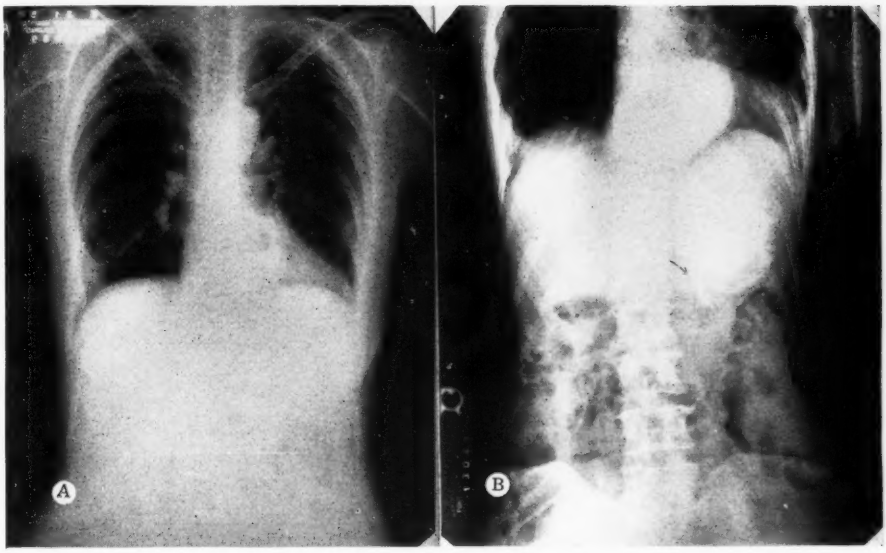


Fig. 2.—Left, posteroanterior chest study showing air fluid level behind the cardiac shadow, suggesting diaphragmatic hernia. Right, another film following barium meal confirms the hiatus hernia noted on the chest plate. Acute organoaxial volvulus with torsion in the distal one-third of the stomach, producing complete obstruction. Actual twisting at the site of the volvulus can be demonstrated (arrow). The greater curvature lies to the right side.

Two weeks after this operation, the patient was seized with a sudden excruciating and expanding pain which was most severe in the right chest but was referred to the epigastrium and both shoulders. She felt that emptying her stomach would be helpful, but she was unable to vomit. Attempts to pass a stomach tube were unsuccessful.

A posteroanterior chest study (Fig. 2, left) showed an air fluid level behind the cardiac shadow suggesting diaphragmatic hernia. After a barium meal (Fig. 2, right), the presence of a hiatus hernia noted on the chest plate was confirmed. There was also an acute organoaxial volvulus with torsion in the distal one-third of the stomach producing complete obstruction. The actual twisting at the site of the volvulus could be demonstrated. The greater curvature lay to the right side.

When seen, this patient was markedly dehydrated. Her thoracic and abdominal cavities were explored through a left thoracotomy incision. A large hiatus hernia sac was opened, the inverted stomach and part of the omentum were reduced, the hiatus hernia was repaired, and the diaphragm and thorax were closed.

This patient recovered and has remained well.

Comment.—To our knowledge, obstructing organoaxial volvulus of the stomach has not been described as a postoperative complication. The exact mechanism of the torsion under these circumstances is not clear. Both cases were associated with hiatus hernia, a condition which frequently accompanies gastric volvulus.

CASE 3.—A 45-year-old woman gave a history of intermittent attacks of excruciating epigastric pain accompanied first by some retching, then by vomiting, which would invariably relieve her acute symptoms. As the patient lived in the country, she would always be asymptomatic by the time she reached a physician. On the last episode she was admitted to the hospital.

X-ray examination (Fig. 3, left) revealed acute total organoaxial volvulus associated with eventration of the diaphragm resulting in high-grade obstruction at the pylorus. The high position of the pylorus was noted. The greater curvature lay superior to the lesser curvature. A 23-hour retention study demonstrated the greater portion of the barium meal still to be in the stomach (Fig. 3, right).

By this time the patient was able to vomit, and her symptoms were relieved. Because the disease was chronic and intermittent, the eventration of the diaphragm was repaired.

The patient has been followed for one and a half years and has had no recurrence or digestive complaints.

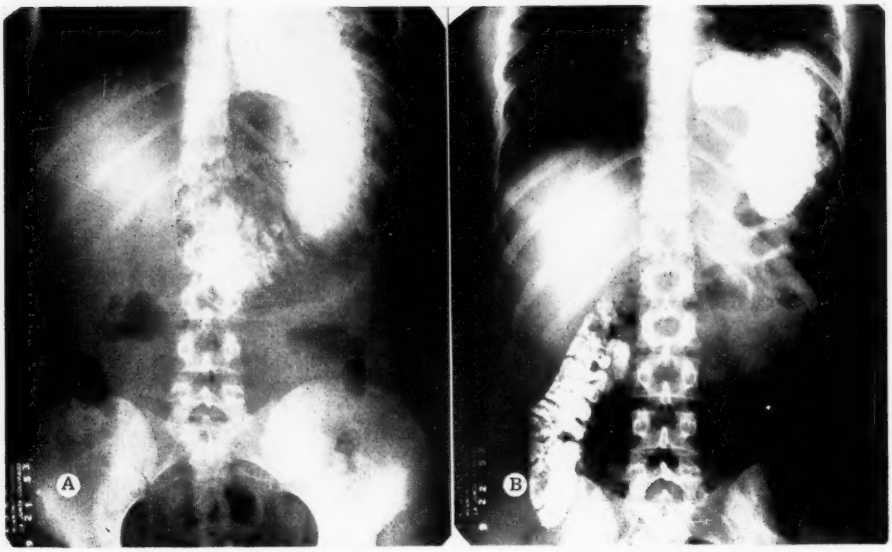


Fig. 3.—Acute, total organoaxial volvulus associated with eventration of the diaphragm, resulting in high-grade obstruction at the pylorus. Left, note high position of the pylorus. The greater curvature lies superior. Right, 23-hour retention study demonstrates the greater portion of the barium meal remaining in the stomach.

Comment.—This case is an example of gastric volvolus associated with eventration of the diaphragm. X-ray differentiation between eventration and diaphragmatic hernia is frequently difficult and sometimes impossible. Preoperatively, it was impossible in this case to identify the diaphragm.

Case 4.—A 62-year-old woman complained intermittently of epigastric pressure and pain which was especially severe after meals. She experienced a great deal of flatulence and belching. These attacks would be relieved either by induced or spontaneous vomiting.

X-ray examination showed a total organoaxial volvulus, which was idiopathic in type. The supine studies (Fig. 4, left) demonstrated the greater curvature uppermost, adjacent to the diaphragm, and extending to the right across the midline of the abdomen. The erect lateral study (Fig. 4, right) demonstrated two fluid levels as well as a low esophageal-gastric junction, differentiating this from a cascade stomach.

At laparotomy, the volvulus was found, but there was no demonstrable etiologic factor. The low-grade volvulus was easily reduced, but it tended to assume its former position. A posterior gastroenterostomy with suture of the stomach to the base of the transverse mesocolon was deemed a suitable procedure for fixation of the malrotated stomach. The patient's postoperative course was complicated by bleeding from the gastrojejunostomy stoma which required correction, but she has remained well for 15 months since this procedure.

Comment.—This is the only idiopathic gastric volvulus case in our series.

It has been emphasized in the literature [11] that volvulus of the stomach up to 180 degrees may occur without obstruction or strangulation of the blood supply and may undergo spontaneous resolution. This patient's history and roentgen findings would indicate such a situation. Rotation beyond 180 degrees produces complete obstruction with likely strangulation, if not corrected.

Case 5.—A 56-year-old woman who had been a cardiac problem for some time complained of substernal pressure and dyspnea after eating. She had developed intermittent burning, epigastric pain, and dysphagia with regard to solid foods. A classical organoaxial volvulus with a diaphragmatic hernia was demonstrated on x-ray studies (Fig. 5). The greater curvature of the stomach was uppermost. The volvulus at the pylorus was producing a partial obstruction.

The patient's symptoms were controlled on a medical program.

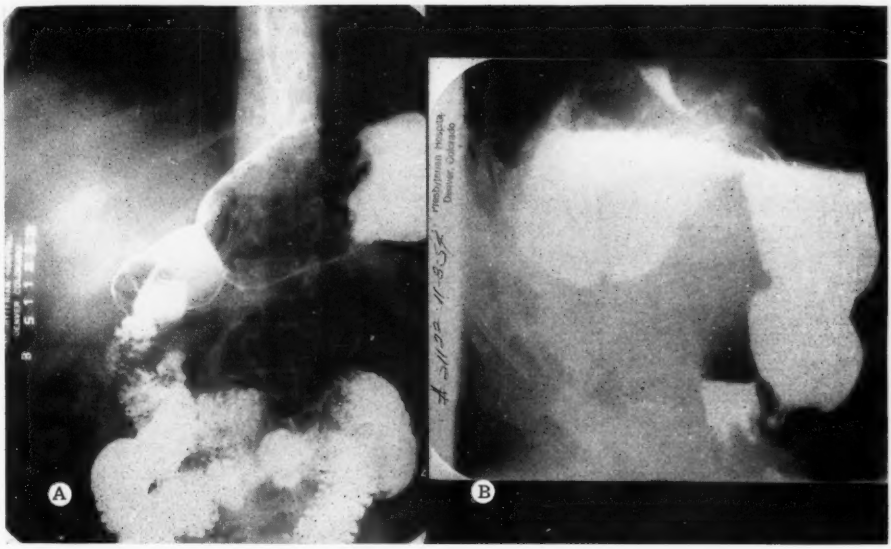


Fig. 4.—Total organoaxial volvulus, idiopathic. Left, supine study demonstrates the greater curvature uppermost adjacent to the diaphragm. Right, erect lateral study demonstrates two fluid levels, as well as low esophagogastric junction, differentiating this from cascade stomach.

Comment. — A conservative course was thought advisable in this instance because of the patient's poor cardiac status, even though obstruction due to the gastric volvulus was considerable.

CASE 6.—A 67-year-old woman who had been under treatment for arteriosclerotic heart disease was admitted to the hospital because of nausea of seven days' duration and vomiting of a mucoid material.

Physical examination revealed evidence of heart-failure and the signs of a small cerebrovascular accident, as manifested by weakness of the left lid, drooping of the left side of the mouth, and a deviation of the tongue to the left. During the next two days in the hospital, the patient was unable to retain any fluids.

An upper gastrointestinal series showed an organoaxial volvulus associated with a hiatus hernia (Fig. 6). The volvulus was through the pars media of the stomach, producing high-grade obstruction. After 10 hours, only a small amount of barium meal passed beyond the site of the volvulus.

The patient's symptoms became progressively severer, and she developed considerable pain. It was thought best to risk an attempted repair of the diaphragmatic hernia.

The thorax was entered through a left posterolateral incision involving excision of the eighth rib. The volvulus was reduced, and the incarcerated stomach was freed and placed beneath the diaphragm. The diaphragmatic crura were sutured posterior to the esophagus with four sutures of

Fig. 5.—Organoaxial volvulus associated with diaphragmatic hernia. The greater curvature of the stomach is uppermost. A volvulus at the pylorus was producing partial obstruction.

Fig. 6.—Organoaxial volvulus associated with hiatus hernia. Volvulus through the pars media of the stomach, producing high-grade obstruction. After 10 hours, only a small amount of barium meal passed beyond the site of the volvulus.

heavy silk. A drain was placed in the 10th interspace, and the thorax was closed.

The patient's condition deteriorated, and she died 36 hours after the operation.

Comment.—This case demonstrates an obstructing volvulus of a stomach incarcerated in a hiatus hernia. Rotation of more than 180 degrees had occurred through the region of the pars media of the stomach, producing obstruction.

Case 7.—A 28-year-old man was admitted to the hospital for an injury which occurred when he fell about 40 feet from a roof to the ground. On admission, he was in semishock and complained of severe pain in the left shoulder. His history was negative, except for a chest injury in 1946. He had been hospitalized for it, and the diagnosis had been fractures of the ribs of the left thorax. His convalescence from this episode was uneventful.

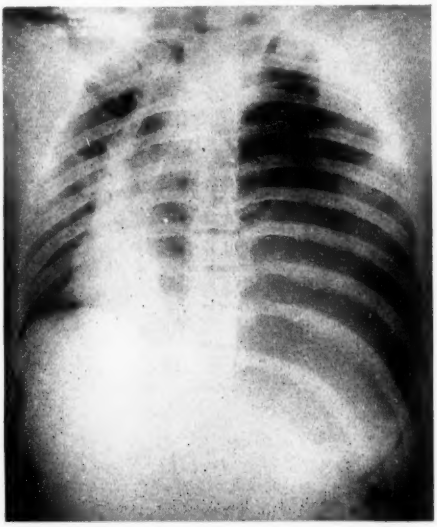
Physical examination was negative, except that the patient held himself rigid. There was a swelling, discoloration, and an effusion of the left shoulder joint. The abdomen was somewhat rigid.

X-ray examination of the shoulder showed a complete comminuted fracture of the upper one-fourth of the left humerus and the upper major fragment displaced laterally about 1 cm. The x-ray examination of the chest at that time showed some evidence of fracture through the callus of the previous fractures of the seventh and eighth ribs in the left thorax. The left hemidiaphragm was only slightly elevated, and the mediastinum appeared to be in normal position. The fracture of the left humerus was reduced and placed in external fixation with a Thomas arm splint.

Twelve hours after admission, the patient, who had been doing well, vomited a small amount of clear fluid, complained of excruciating pain in his chest, and went into severe shock. He died approximately three hours later.

A postmortem chest roentgenogram (Fig. 7) showed an acute gastric volvulus with hernia of the entire stomach above the diaphragm, pneumothorax, and dilatation of the stomach, which filled the entire left hemithorax. The entire left lung was collapsed and the mediastinum shifted to the right. Permission for autopsy was obtained, and the autopsy was performed. The chest was opened by an incision extending through the sternal notch to midway between the umbilicus and the xiphoid. When the pleural cavity on the left was entered, there was a distinct escape of air. The anterior thoracic wall, thus mobilized was reflected superiorly. The mediastinum was displaced greatly toward the right, and the stomach displaced the left lung, which was pressed tightly against the medial wall of the left pleural cavity. The entire pleural cavity was filled with the distended stomach and a portion of the transverse colon. There was a small amount of serosanginous fluid in the pleural

Fig. 7.—Postmortem chest roentgenogram. Acute gastric volvulus. Postraumatic hernia of the entire stomach above the diaphragm. Dilatation of the stomach which fills the entire left hemithorax. Volvulus at the pylorus, producing obstruction.



cavity. Examination of the diaphragm revealed a 2-in. laceration of the left dome. This laceration was 2 in. lateral to the hiatus for the esophagus. There was a complete torsion of the pyloric end of the stomach.

The pathologist's diagnosis was cardiac tamponade due to herniation and strangulation of the stomach in the left pleural cavity through a laceration of the dome of the diaphragm and simple, comminuted fractures of the shaft and the head of the humerus on the left.

Comment. — Incarceration of organs through a traumatic tear in the diaphragm is not uncommon; however, marked distention of a herniated stomach, secondary to an obstructing volvulus in the pylorus, is considered an exceedingly rare event.

GENERAL COMMENT

Berti [4] reported the first case of gastric volvulus in 1866. Berg [3] reputedly performed the first operation for relieving gastric volvulus in 1895. In 1909, Payer [9] found 22 cases of acute gastric volvulus in the literature and added 12 cases. In 1914, Kocker [8] found 28 cases and added one more. In 1921, Deaver and Ashhurst [6] said that volvulus of the stomach had apparently been observed in 35 instances and that there were on record at least 22 cases in which operation had been performed. S. N. Sennett [10] stated that Nikos and Anagostides found a total of 116 cases reported up to 1940. Since then, Bazzano and Hood [2] have found 76 additional reported cases and have added another. Gottlieb [7] and associates added 20 cases and an excellent review of the subject.

Many classifications of gastric volvulus have been proposed, and many anatomic variations have been described. All are helpful, but none is complete. Anzilotti [1] divided volvulus into acute, subacute, and intermittent. Payer [9] based his classification, which is reproduced here, upon etiology.

1. Idiopathic volvulus
2. Secondary volvulus
   a. Hernia or eventration of the diaphragm
   b. Tumors of the stomach
   c. Inflammatory processes
   d. Peptic ulcer and/or hourglass stomach
   e. Postoperative conditions
   f. Displacement by neighboring organs, including the gravid uterus

Von Haberer [13] has forwarded a topographic classification, which is reproduced as the most satisfactory to date.

I. Type:

(a) Organoaxial—Rotation of the stomach upward around the long axis of the stomach, i. e., around the coronal plane

1. Supracolic
2. Infracolic

(b) Mesenteroaxial—Rotation of the stomach from right to left, or left to right about the long axis of the gastrohepatic omentum (radix mesenterii)

II. Extent:

(a) Total—Cases in which the whole stomach except the diaphragmatic attachment rotates

(b) Partial—Instances in which the rotation is limited to a segment of stomach, usually the pyloric end

III. Direction:

(a) Anterior—Cases in which the rotating part passes forward

(b) Posterior—Cases in which the rotating part passes backward

IV. Etiology:

(a) Secondary to disease in the stomach or adjoining organs (see Payer's classification)

(b) Idiopathic

V. Severity:

(a) Acute—Presenting a picture of an acute abdomen

(b) Chronic—Causing constant or recurrent milder symptoms, or symptomless

In attempting to understand the mechanism of torsion of the stomach, one must remember that the stomach is held firmly only by the cardia and that the pylorus is not so firmly fixed. When a torsion occurs, the pylorus moves closer to the cardia. The greater curvature, in most cases, moves anteriorly and upward until it lies beneath the left portion of the diaphragm. The mesocolon is stretched and drawn upward so that the colon comes to lie flattened across the posterior surface of the stomach in organoaxial rotation.

In mesenteroaxial torsion the proximal portion of the stomach falls on the distal portion, or the distal portion falls on the proximal portion, and the axis is somewhere in the central portion. Some observers,[2] at autopsy, attempted to determine in a number of cadavers with normal stomachs if the stomach could be forced into volvulus. They noted that unless the gastrocolic ligament was extremely short, it was easy to rotate the stomach upward (organoaxial rotation). In only one cadaver was it possible to rotate the stomach on its mesenteric axis 180 degrees, since the duodenal and the cardiac attachments in the others were too well fixed.

Etiology.—Bazzano and Hood [2] attribute acute volvulus to (*a*) relaxed gastrohepatic, lienal, and colic ligaments; (*b*) generalized ptosis, and (*c*) faulty peritoneal attachment of the duodenum. They assume that any of these conditions may allow excessive mobility of the stomach. Chronic volvulus, often called intermittent, partial, or complete, is frequently due to some extrinsic or intrinsic lesion of the stomach. In most such cases, some congenital or acquired anomaly is present. Diaphragmatic hernia, tumors, peptic ulcer, or displacement of the stomach are common offenders. Gastritis, overfilling of the stomach, vomiting, and gaseous distention are usually predisposing causes. Some cases occur in the early postoperative period, as was demonstrated in Cases 1 and 2 in our series. Six of the seven cases in our series were secondary to either a congenital or an acquired defect in the diaphragm.

Pathology.—The pathologic change occasioned by volvulus of the stomach depends upon the degree of rotation of the stomach and on the length of time that it has remained in this abnormal position. In acute cases twisting causes closure of the pylorus and later the cardia. The stomach becomes greatly distended, edematous in some cases, and thinned out or stretched in others. The blood supply is in jeopardy. Cases of necrosis with rupture are not uncommon. Because the viscus cannot empty itself, a closed cardia prevents vomiting and also renders gastric intubation impossible.[2] The peritoneal cavity may be full of clear exudate or of gastric contents if the stomach has ruptured. The colon may be extremely distended in the supracolic, organoaxial type, or it may be empty in the mesenteroaxial type.

Symptoms.—The symptomatology in acute gastric volvulus is often dramatic and variable, depending upon whether the volvulus is acute, subacute, or chronic. In the acute complete cases the onset is sudden. The pain is usually described as expanding and as starting in the epigastric region. At first, the patient vomits, then the vomiting turns into an unproductive retching. The systemic signs manifest themselves early in the course of the disease. The patient develops shock-like symptoms, becomes pale, and perspires; his blood pressure drops, and he usually has some fever. The abdomen becomes markedly distended and tender. These symptoms may be mild, acute, or intermittent. Toygar [12] stresses Borchardt's and Lenormant's triad, consisting of strong efforts to vomit without results, circumscribed epigastric pain, and the impossibility of passing a stomach tube. This triad was present in all acute cases which we observed. We believe it to be of great value in making one suspect the diagnosis of gastric volvulus.

Differential Diagnosis.—A few of the more important conditions confused with acute gastric volvulus are ruptured peptic ulcer, intestinal obstruction, abdominal apoplexy, acute pancreatitis, and mesenteric embolism. Chronic gastric volvulus is most apt to be confused with gastritis, peptic ulcer, and gallbladder disease.

The roentgenologic diagnosis depends upon the recognition of malposition of the curvatures of the stomach and identification of the actual site of torsion. In organoaxial volvulus the greater curvature lies superior to the lesser curvature owing to the forward and upward rotation of the stomach. As rotation continues, the greater curvature lies superior and to the right, and the pylorus frequently is rotated upward and to the left.

In acute volvulus the actual torsion or twist can sometimes be visualized. When obstruction is complete, recognition of malrotation is essential to make the diagnosis. Volvulus should be considered in cases of obstruction of the stomach when diaphragmatic hernia or eventration of the diaphragm is present.

The presence of two fluid levels, low position of esophagogastric junction, and malposition of greater curvature differentiates it from cascade stomach.

Treatment.—In many cases of chronic or intermittent volvulus, conservative therapy, as was used in Case 5, undoubtedly suffices. In persons in whom symptoms persist and are unbearable and in all those with acute obstructive conditions, some type of operative intervention is imperative. The procedure employed depends upon the etiology of the volvulus and upon the condition of the stomach and the contiguous structures at the time of operation. Numerous authors have said that in many cases of primary volvulus they had only to restore the stomach to its natural position in the abdomen and to guide a tube into it. In the only patient with idiopathic volvulus in our series, the stomach appeared so mobile and the likelihood of recurrent volvulus so imminent that we thought a fixation procedure was indicated. Placing a row of sutures from the anterior stomach wall to the anterior peritoneum and performing a posterior gastrojejunostomy have both been suggested. Neither seemed too physiologic, but we felt that, of the two, posterior gastrojejunostomy would be easier to revise, if necessary, and less likely to produce symptoms from other mechanical complications. This procedure was successful in Case 4.

Secondary gastric volvulus presents some other organic lesion, such as ulcers, adhesions, tumors, or herniation, as a significant causative factor. In treating it, a procedure designed to correct the anomaly causing the volvulus is essential. Bazzano and Hood [2] stressed the importance of keeping these corrective measures to a minimum and of reserving extensive resection, if it might be necessary, for a second procedure, when the patient's general condition might be improved. Reduction of the volvulus with repair of an existing hiatus hernia sufficed in Cases 1 and 2. Repair of an eventration of the diaphragm resulted in a cure in Case 3. In Case 1 the stomach appeared so mobile and edematous that we thought the patient would have difficulty of some kind; so the hiatus hernia was repaired and a subtotal gastrectomy was performed. The procedure was successful, and the patient has remained well.

SUMMARY

Seven cases of gastric volvulus, in six of which the clinical and the roentgenologic picture was that of acute obstruction, are reported.

Six of the seven cases were associated with a congenital or an acquired defect in the diaphragm. Immediate operative intervention was indicated in five of the seven cases. Obstructing gastric volvulus proved to be a postoperative complication in two of the seven cases. One case of chronic idiopathic volvulus was operated upon because of the persistence of symptoms.

One case, of necessity, was managed on a medical regime.

DISCUSSION

DR. R. MORTON BOLMAN, Fort Wayne, Ind.: I certainly have enjoyed this paper, and I arise merely to say that we have had two such cases in a period of four weeks, never having seen one before.

On that basis it would certainly seem possible that these are commoner than one would suspect. One of our cases was accompanied by a foramen of Bochdalek hernia, the other a malrotation of the small intestine with a paraesophageal hiatus hernia. The first diagnosis was made by giving the patient gall-bladder dye, which demonstrated the relative position of the pylorus and the cardia and made the diagnosis possible.

REFERENCES

1. Anzilotti, A.: Sul volvolo gastrico (Studio patogenetico clinico e radiologico), Arch. ital chir. **26**:1-49, 1930.

2. Bazzano, J. J., and Hood, T. K.: Volvulus of the Stomach: Report of a Case and Review of the Literature, Ann. Surg. **135**:415-420, 1952.

3. Berg, J.: Zwei Fälle von Achsendrehung des Magens, Nord. med. Ark. **19**:1-18, 1897.

4. Berti, A.: Singulari attortigliamento dell'esofago col duodeno seguito da rapida morte, Gazz. med. ital. **9**:139, 1866.

5. Borchardt, M.: Zur Pathologie und Therapie des Magenvolvulus, Arch. klin. Chir. **74**:243-260, 1904.

6. Deaver, J. B., and Ashhurst, A. P. C.: Surgery of the Upper Abdomen, Philadelphia, Blakiston Company, 1921.

7. Gottlieb, C.; Lefferts, D., and Beranbaum, S. L.: Gastric Volvulus, Am. J. Roentgenol. **72**: 609-638, 1954.

8. Kocher, T.: Ein Fall von Magenvolvulus, Deutsche Ztschr. Chir. **127**:591-635, 1914.

9. Payer, A.: Volvulus ventriculi und die Achsendrehung des Magens, Mitt. a. d. Grenzgeb. d. Med. und Chir. **20**:686-726, 1909.

10. Sennett, S. N.: Volvulus of the Stomach, South African M. J. **14**:192-193, 1940.

11. Singleton, A. C.: Chronic Gastric Volvulus, Radiology **34**:53-61, 1940.

12. Toygar, O.: Die Formveränderungen des Magens (Kaskadenmagen, Volvulus des Magens), Schweiz. med. Wchnschr. **78**:767-772, 1948.

13. Von Haberer, H.: Volvulus des Magens bei Carcinom, Deutsche Ztschr. Chir. **115**:497-532, 1912.

Acute Stoneless Cholecystitis

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Most gall bladders surgically removed contain stones. The incidence of cholelithiasis in published cholecystectomy series ranges from 75% to 100%, with an average for the aggregate of 92.5%.[1] Only occasionally has stone-free cholecystitis been considered separately.* However, acute inflammation, and even gangrene, of the gall bladder can occur in the absence of calculi. The pathogenesis of this form of cholecystitis is often obscure, since acute cholecystitis is generally attributed to blockage of the cystic duct by a gallstone.

The present study was undertaken in a search for clues to the etiology of acute stoneless cholecystitis and to emphasize certain features apparently peculiar to this form of gall-bladder disease. The impression gained is not in accord with the oft-quoted statement that "cholecystitis without stones seems to belong to a region on the borderline between functional and organic disease." [2]

MATERIAL

A total of 542 cholecystectomies were performed during the five years ending August, 1954 (Table 1). Gallstones were found in 94.1% of the cases. In 13 patients the pathological report suggested that the gall bladder was essentially normal. Some of these cholecystectomies had been done because of recurrent pancreatitis. In five instances there was significant fibrosis and thickening of the gall-bladder wall but nothing clinically or pathologically to suggest an acute cholecystitis. Fourteen patients with histologically confirmed cholecystitis in the absence of stones make up the material for this report (Table 2).

Read at the 63d Annual Meeting of the Western Surgical Association, Seattle, Dec. 1, 1955.

From the Departments of Medicine and Surgery, The Mason Clinic, and The Virginia Mason Hospital.

* References 2 and 3.

OBSERVATIONS

Gangrenous Cholecystitis.—Of the 14 patients with acute stoneless cholecystitis, 5 had gangrene of the gall bladder. Four were men and all but one were more than 70 years of age (Table 3). None had prior symptoms to suggest gall-bladder dysfunction. The acute illness was sudden in onset and characterized by severe, steady epigastric pain, nausea, and vomiting. The clinical condition deteriorated progressively over the 12 to 96 hours between onset and admission to the hospital, except for the 76-year-old man, who seemed to improve after the first day, permitting a delay of 10 days before surgery was performed. In three cases the epigastric pain settled into the right upper quadrant, but in the other two the severer pain was in the

TABLE 1.—*Cholecystectomies Performed at the Virginia Mason Hospital, August, 1949-August, 1954*

	Number of Cases	Per Cent
Gallstones present	510	94.1
No gallstones found	32	5.9
Total	542	100.0

TABLE 2.—*Stoneless Cholecystitis Cases*

Type	Number
Gangrenous	5
Simple acute	9
"Chronic"	5
Equivocal	13
Total	32

TABLE 3.—*Gangrenous Cholecystitis Without Gallstones*

Sex	Age, Yr.	Preoperative Diagnosis
M	65	Acute cholecystitis
M	72	Acute appendicitis
M	76	Resolving cholecystitis
M	82	Acute appendicitis
F	82	Empyema gall bladder
	Average 75.4	

lower right part of the abdomen. All of the patients were febrile upon entry to the hospital with oral temperatures ranging from 99.6 to 102 F. Two were in shock and two had detectable slight scleral icterus.

Abdominal tenderness was generalized in one case but confined to the right side of the abdomen in the other four. However, the two patients whose pain centered in the lower right quadrant were also most tender in that region. Muscle spasm was minimal in all patients. The gall bladder was palpable in one, a patient with torsion of the gall bladder.

Leucocyte counts ranged from 12,800 to 21,200, with from 75% to 96% neutrophiles. Amylase content of the blood was tested and found normal in all instances. A trace to 2+ (basis of 3) albumin was present in the urine of all patients. Cholecystography was not carried out in three instances, had shown a normal stone-free gall bladder two years previously in one patient, and had shown a nonfunctioning viscus in the fifth. The last-mentioned was the patient who appeared to be recovering from his acute illness, permitting cholecystography to be done.

The preoperative diagnosis was clearly erroneous in three patients (Table 3).

At surgery an obviously gangrenous gall bladder was found in each case, with perforation and pericholecystic abscess formation in two. Torsion of the gall bladder had occurred in one patient, leading to gangrene of the distal half. The cystic and common bile ducts were normal to inspection and palpation in all patients. Culture of gall-bladder contents disclosed Escherichia coli in one, no growth in two, and was not obtained in two. Amylase content of gall-bladder bile was determined only once and found to be 1400 units.

Pathological examination of the gall bladders failed to disclose stones or gravel in any case, and there was no suggestion of previous inflammatory process. The postoperative course was uneventful in four cases. The 82-year-old man succumbed on the second postoperative day with continuing shock, despite supportive measures. Follow-up information is available for the other four patients, all of whom were doing well when last examined two months to two years postoperatively.

As is apparent from the preoperative diagnoses, gangrenous cholecystitis without cholelithiasis can present a misleading clinical picture. The lack of previous colic, jaundice, excessive gaseousness, or fatty-food dyscrasias deprived the clinicians of any leads to the underlying pathology. In one patient a cholecystogram obtained two years earlier during the course of a general medical survey tended to shift suspicion away from the gall bladder. The two patients whose principal pain and tenderness were low on the right side of the abdomen were both thought to have appendicitis. In one case the palpable gall bladder led to a diagnosis of cholecystic disease, although torsion of the gall bladder was unsuspected. The two patients with slight scleral icterus were both diagnosed correctly, and, although preoperatively common-duct stones were suspected, none were found, and their postoperative course gave no indication that stones had been overlooked. Liver biopsy in one showed cholangitis, which probably accounted for the jaundice.

Patients with gangrene of the gall bladder tend to be older than those with simple acute cholecystitis. The ages of these five patients is particularly impressive (average age 75.4 years) when compared with the remainder of the group (average age 44.0 years). Men are better represented, both in series of stoneless cholecystitis and in series with gangrene whether or not stones are present. This was true of our group, four of the five being men. The one woman had torsion of the distal half as the etiologic factor of the gangrene. In this regard it is interesting to note that Short and Paul [4] found that of a series of 53 cases of torsion of the gall bladder leading to gangrene, all but 6 were female. These authors noted that torsion of the gall bladder was seldom diagnosed preoperatively but suggested that a gall bladder palpable with varying degrees of ease from time to time may be indicative.

The patient who seemed by all clinical and laboratory measures to be recovering from

an uncomplicated acute cholecystitis but who was found to have a gangrenous gall bladder remains an enigma. That elderly people can conceal considerable acute intra-abdominal pathology from the clinician is well known. This single case would suggest that conservative management of acute cholecystitis might be hazardous in older patients.

Acute Cholecystitis.—The clinical picture presented by the nine patients with acute stoneless cholecystitis but without gangrene more nearly fulfilled the common concept of a typical gall-bladder patient. Six were women and the average age was 44 years with little spread (Table 4). However, they were not flatulent or dyspeptic, and none had complained of excessive gaseousness or any notable degree of fatty-food intolerance. None had suffered colicky right upper quadrant pains in the past, and only one had had a previous unexplained attack of indigestion of any severity. The presenting illness apparently was the initial episode of cholecystitis in all of the others. In four cases the duration of the illness was brief, that is, less than two days before the patient entered the hospital. The other five patients had been ill for from several days to two weeks. Common to all was the initial symptom of steady abdominal pain, confined to the right upper quadrant in six but beginning in the epigastrium in three. The only patient with colicky pains during the presenting illness was subsequently found to harbor an adult Ascaris lumbricoides roundworm in the common duct. Four patients vomited and two were mildly febrile upon entry to the hospital.

TABLE 4.—*Acute Nongangrenous Cholecystitis Without Stones*

Sex—3 males and 6 females	
Average age—44 years	
Past history—negative	
Present illness—the first attack	
Complications	
Empyema gallbladder	1
Ascariasis	1
Giardiasis	2
Diagnosis—accurate	

The 29-year-old man had experienced several attacks of acute epididymitis, type unknown, during the year prior to the development of acute cholecystitis. Giardia lamblia had been found in large numbers in his stool a week before the presenting illness and quinacrine (Atabrine) hydrochloride therapy was given. Upon entry to the hospital he was acutely ill, with board-like rigidity of the abdomen, temperature orally of 102.3 F, and a leucocyte count of 24,750. This rose to 50,750 with 90% polymorphonuclear leucocytes and no eosinophilia. On the second day, the gall bladder became palpable and surgery was performed. A markedly distended gall bladder was found with normal cystic and common ducts. The pancreatic head was negative to palpation. Culture of gall-bladder contents was negative. Convalescence was uneventful, but a mild epididymitis recurred on one occasion several months later. One additional patient also had Giardia lamblia in the stool.

Physical examination of the remaining patients revealed moderate to severe tenderness limited to the right upper quadrant in each case. None was jaundiced. The gall bladder was palpated in one patient who was found at surgery to have developed an empyema of the gall bladder. Leucocyte counts determined for six patients were but mildly elevated from 10,800 to 16,600 with an average count of 12,400 per cu. mm. Differential leucocyte count, done in four cases, revealed more than 65% neutrophiles in three.

Cholecystography was performed in six cases and revealed a nonfunctioning gall bladder in five. The young man with epididymitis had had a cholecystogram done five months previously. An extremely large gall bladder was visualized. Serum amylase determinations, done in five patients, was elevated in one, the patient with Ascariasis. The preoperative impression was correct in every case: acute cholecystitis, eight cases; empyema of the gall bladder, one case. However, the presence of Ascariasis was unsuspected, although subsequent stool studies revealed Ascaris ova.

At surgery the gall bladder was described as inflamed in each patient, distended in four, and contracted in one. None had per-

forated. The cystic duct was normal to palpation and inspection in all but one patient in whom a partial stricture was found just proximal to the common duct. An enlarged (but not biopsied) lymph node was described as lying adjacent to the junction of the cystic and common ducts in one patient. The only abnormal common duct was the one harboring an adult Ascaris worm. The others were judged normal to inspection and palpation. Operative cholangiogram was performed but once and was normal. The pancreas was normal to palpation in the seven cases where mention of this organ was made in the operative report. Culture of gall-bladder contents was negative in the two cases studied. The test for amylase in the gall-bladder bile was not carried out.

The pathological diagnosis was acute cholecystitis, six cases (one with Ascariasis as well); resolving acute cholecystitis, two cases, and empyema of the gall bladder, one case. Two of the patients with acute cholecystitis may have had some previous inflammation of the gall bladder, as judged by mild fibrosis of the gall-bladder wall.

Diagnosis was not difficult in these patients with simple acute cholecystitis without stones. In retrospect, one wonders if perhaps the absence of previous symptoms of gall-bladder dysfunction might have given pause. The experience with these patients emphasizes that, like the appendix, the gall bladder can cause an acute illness without prior warning. However, the combination of abdominal pain settling in the upper right quadrant plus definite tenderness to palpation of this area apparently was decisive enough to permit correct preoperative diagnosis. Of course, the finding of a palpable gall bladder or a nonfunctioning gall bladder by cholecystography gave valuable confirmation.

Follow-up information was available for three patients in this group. In none did symptoms or signs develop to suggest that choledocholithiasis had been overlooked. None of the nine patients suffered complications while in the hospital, and there were no deaths.

The single instance of Ascaris blockage of the common duct recalls to mind an oft-mentioned but seldom-encountered complication of Ascaris infestation. The elevated blood amylase in that patient was evidence for pancreatic as well as cholecystic involvement, probably due to duct blockage. Giardia lamblia has been suspected of causing cholecystitis and cholangitis.[5] Unfortunately, gall-bladder contents were not examined for parasites in the cases presented.

Chronic Stoneless Cholecystitis.—Five patients were found to have histologic evidence of extensive fibrosis and thickening of the gall-bladder wall without stones or acute cholecystitis. All had been troubled with recurrent indigestion and gaseousness in the past. In this respect these patients contrasted sharply with the 14 cases of acute or gangrenous cholecystitis mentioned before. One patient, a 54-year-old man, had transient jaundice associated with severe abdominal pains one week prior to admission to the hospital. He may have passed a common-duct stone, but no stones were present at the time of surgery. The common duct was explored and irrigated and a cholangiogram done, with negative results. A 67-year-old man was found to have a deep transverse cleft which divided the gall bladder into two sections, the distal being filled with clear bile. A 37-year-old woman had cholecystitis glandularis, chronic, with intramural calculus formation but no free stones in the lumen of the gall bladder or in the ductal system. A 27-year-old woman had fatty-food dyscrasia, gas, and epigastric distress without colic or jaundice for four years following a pregnancy. The gall bladder was markedly thickened, and histologic study revealed fibrosis and diffuse chronic inflammatory infiltrate, but no stones were present.

Cholecystography in these patients revealed a nonfunctioning gall bladder without evidence of opaque gallstones. In the fifth case a calcified gall-bladder wall was visualized.

The preoperative impression was chronic cholecystitis in each case. The cystic and

common ducts were normal to inspection and palpation in all five patients. No abnormality of the pancreas was described. All of these patients were relieved of their chronic digestive symptoms by cholecystectomy.

COMMENT

The present study confirms the relative infrequency of stoneless cholecystitis. The 94.1% incidence of gallstones among the 542 gall bladders surgically removed is in accord with the average incidence of 92.5% reported by Berk [1] for 17 large series published between 1930 and 1940. Cholecystitis without cholelithiasis is obviously of little importance statistically, a fact which probably explains as well as justifies the preponderant attention which has been paid the combined disorder. However, we feel that it is useful to give heed occasionally to the less common and, hence, atypical forms of clinical disease processes.

Patients with acute stoneless cholecystitis could be likened to victims of acute appendicitis or pancreatitis in that the presenting illness was unheralded. Sudden departure from previous good health and digestion was particularly striking. Previous acute attacks of colic, indigestion, flatulent dyspepsia, or fatty-food dyscrasia were notably absent.

Physical findings were misleading only in the gangrenous form in which the abdominal pain and tenderness did not always center in the gall-bladder area. Helpful but seldom encountered physical signs included a palpable gall bladder, when torsion or empyema of the viscus had occurred, and scleral icterus due to associated cholangitis.

In the nongangrenous forms of stoneless cholecystitis in which time for cholecystography was available, x-ray examination proved generally helpful.

Etiology.—Why should a patient without gallstones develop acute cholecystitis or gangrene of the gall bladder? The generally accepted cause of acute inflammation of the gall bladder is blockage of the cystic duct. Womack and Bricker,[6] in a discussion of the pathogenesis of cholecystitis stated, "Acute cholecystitis unassociated with obstruction of the cystic duct is extremely rare." The cause of the blockage is almost always a gallstone. When acute inflammation of the gall bladder develops in the absence of stones, other causes must be sought. Rare instances of cystic-duct obstruction due to stricture, adjacent lymphadenopathy, apparent kinking of a redundant duct, or torsion with closure of the duct have been referred to in the literature.[7] In the present series one patient with acute cholecystitis had a partial stricture of the cystic duct, and in another an enlarged lymph node was present adjacent to the duct; but this is a rather common finding. The pathogenesis of the remaining instances of acute cholecystitis cannot be explained on the basis of cystic-duct blockage.

Another factor which has been suggested as causing acute cholecystitis is infection: hematogenous, or by extension from adjacent viscera, or from the duodenum by way of the biliary tree. Moynihan [8] considered bacterial infection to be of considerable importance in causing cholecystitis. Various other investigators have felt similarly. However, more recent studies have tended to discount the infectious factor,[9] although the gall bladder is involved by typhoid and other septicemias. An example of acute suppurative staphylococcic cholecystitis without cholelithiasis following recurrent furunculosis has been reported from this clinic.[10] Cultures were taken of the gall bladder in only four cases in the present series. In one E. coli was recovered, whereas the other three cultures were sterile. In two cases, not cultured, pathological evidence of bacterial colonization in the gall-bladder wall was noted. Thus, it would seem that infection might play a role only in some instances of acute stoneless cholecystitis.

Wolfson and Rothenberg [3] noted that an infectious process elsewhere in the body had preceded the development of acute cholecystitis in 9 of their 31 cases of acute noncalculous cholecystitis. These authors regarded hematogenous infection a frequent cause of acute cholecystitis. Preceding infection, an epididymitis had occurred in only one of our patients. Although specific questioning on this point was probably not done, it is doubt-

ful whether a recent febrile illness of any significance would have been overlooked in the anamnesis.

Chemical irritation of the gall bladder has also been suggested as a cause of acute cholecystitis. Bile salts and pancreatic enzymes have been suspected. Experimental studies on dogs disclose that either is capable of inflaming the gall bladder.[9] However, Popper [11] found elevated amylase concentration in the gall-bladder bile of only 10% of 200 patients with acute cholecystitis. Unfortunately, gall-bladder amylase was determined in only one of our cases and was not elevated beyond the level often encountered in chronic stone-bearing gall bladders.

Other factors considered but apparently not active were dietary indiscretion, prolonged fasting, dehydration, overexertion, trauma, and recent surgery. The last has recently been called again to the attention of the profession.[12] It is interesting to note that of 17 cases of acute cholecystitis following unrelated surgery, reported by Schwegman and De Muth,[13] stones were not found in 6. The authors remarked, "The high incidence (35 per cent) of calculi-free gallbladders in this series is unexplained." Apparently our patients with acute cholecystitis were suddenly taken ill in the midst of their usual mode of living. Recent changes in activities or dietary habits had not occurred.

The failure to discover a cause for most cases of acute stoneless cholecystitis emphasizes the need for more careful study of the problem. If a patient is found to have an acutely inflamed gall bladder which is free of stones, the surgeon should carefully search for other causes. Congenital anomalies of the cystic duct, such as a partial stricture or other reasons for duct blockage, should be sought. Thrombosis of the cystic artery might be considered and has been encountered by one of us (J. W. B.) prior to this study. The contents of the gall bladder should be cultured as well as tested for pancreatic-enzyme and bile-salt concentration. Parasitic infestation must be considered.

Finally, it should be emphasized that the absence of stones in the gall bladder does not guarantee against their possible presence in the common duct. Two such cases were encountered during the present study but not included in the report. Walters and Snell [14] report a 6% incidence of stone-free gall bladders in patients with ductal lithiasis. The common duct should be explored if any of the usual indications are present.[15]

SUMMARY

The clinical, operative, and pathological records of 14 patients with acute cholecystitis without cholelithiasis are reviewed. Of these five patients had gangrene of the gall bladder.

Patients with acute stoneless cholecystitis do not present the usual picture of gall-bladder dysfunction in that they have no previous digestive symptoms and are notably free of colic.

Patients with gangrene of the gall bladder without stones are often men of advanced age who have had no previous digestive symptoms and who may present misleading physical findings.

The clinical results of cholecystectomy in these patients with stoneless cholecystitis were excellent. Postcholecystectomy symptoms were absent.

In most cases of acute or gangrenous stoneless cholecystitis a precipitating factor cannot be identified and pathogenesis remains obscure.

Suggestions are made for more detailed study of patients with acute stoneless cholecystitis in an attempt to clarify the etiology of this form of gall-bladder disease.

DISCUSSION

DR. CYRIL COSTELLO, St. Louis: In a similar size group of patients in our City Hospital in St. Louis, we have had pretty much the same experience as Dr. Gibbons and Dr. Baker have reported today. Several discrepancies, however, will be evident if we review these findings.

[Slide] This group of patients was approximately the same size, and they were operated on at City Hospital between 1947 and 1955. Twenty-three of these patients had acute cholecystitis without stones.

[Slide] This slide demonstrates the type of operation that was performed. Cholecystostomy in the acute group was performed in 17 cases, while chole-

cystectomy was performed in 6 cases. In the group in which cholecystostomy was performed, two had empyema of the gall bladder; one, acute hydrops; four, acute cholecystitis; six, gangrene; two, pericholecystic abscess, and two had perforations. In the cases in which cholecystectomy was performed, all had acute cholecystitis. I should like to give you a thumbnail sketch of these patients. All the patients with acute cholecystitis were also elderly and all but one of them were men.

[Slide] This was an 85-year-old man who presented himself with constant pain extending to the back of two days' duration. There was a past history of a similar attack three months previously treated at another hospital by "removal of stones." An acute gangrenous gall bladder was found and removed.

[Slide] The next patient was a 74-year-old man who was awakened from his sleep at 2 a. m. by severe pain in the right upper quadrant. He had no previous history of gall-bladder attacks, and the finding of significance was tenderness over the gall bladder.

[Slide] The next patient was a 71-year-old man who was awakened at 3 a. m. by constant severe epigastric pain. He had a past history of mild bloating following meals for one year. Culture of his gall bladder at the time of operation revealed Clostridium welchii.

[Slide] The next patient was our youngest man, 58 years old, awakened from sleep by severe epigastric pain. He had many years' history of typical gall-bladder attacks, and he presented exquisite tenderness over his gall bladder. This patient had a perfectly benign postoperative course.

[Slide] This 72-year-old woman presented herself with constant severe right upper quadrant pain, nausea, and chills for three days. She had had flatulence following meals for one month. She had tenderness over her gall bladder.

All these patients you would recognize as having fairly classical clinical pictures of acute cholecystitis. It was impossible, at the time of operation, to determine before opening the gall bladder whether these patients had stones. I have been able to discover no criterion by which one may be more suspicious of patients who have cholecystitis without calculus.

I believe the paper presented by Dr. Gibbons is a timely one and emphasizes what has been deemphasized frequently in the past, namely, that patients need not have stones to have acute cholecystitis any more than they need have fecaliths in the appendix in order to develop appendicitis.

The relative significance of other etiologic factors certainly remains an enigma, although I believe Dr. Gibbons has summarized all of the theories.

Dr. Thomas B. Gibbons, Seattle: I should like to thank Dr. Costello for his kindness in discussing our paper. I was impressed, hearing his case protocols, with the similarity between his cases and our own.

One point that I should like to mention is that one of the patients mentioned in the paper who did have slight icterus had no common-duct stones, but a liver biopsy revealed evidence of a cholangitis to which we attributed the mild jaundice.

REFERENCES

1. Berk, J. E.: The Management of Acute Cholecystitis, Am. J. Digest. Dis. **7**:325-332 (Aug.) 1940.

2. Mackey, W. A.: Cholecystitis Without Stone: Investigation of 264 Operated Cases from Clinical, Radiological and Pathological Aspects; Attempt to Determine Factors of Service in Estimating Prognosis, Brit. J. Surg. **22**:274-295 (Oct.) 1934.

3. Wolfson, W. L., and Rothenberg, R. E.: Acute Noncalculus Cholecystitis: A Study of 31 Cases, J. A. M. A. **106**:1978-1980 (June 6) 1936.

4. Short, A. R., and Paul, R. G.: Torsion of the Gallbladder, Brit. J. Surg. **22**:301-309 (Oct.) 1934.

5. Hartman, H. R.; Kyser, F. A., and Comfort, M. W.: Infection of the Gallbladder by Giardia Lamblia, J. A. M. A. **118**:608-609 (Feb. 21) 1942.

6. Womack, N. A., and Bricker, E. M.: Pathogenesis of Cholecystitis, Arch. Surg. **44**:658-676 (April) 1942.

7. Cole, W. H.; Novak, M. V., and Hughes, E. O.: Experimental Production of Chronic Cholecystitis by Obstructive Lesions of the Cystic Duct, Ann. Surg. **114**:682-696 (Oct.) 1941.

8. Moynihan, B. G. A.: The Gallbladder and Its Infections, Brit. M. J. **1**:1-6 (Jan. 7) 1928.

9. Gatch, W. D.; Battersby, J. S., and Wakim, K. G.: The Nature and Treatment of Cholecystitis, J. A. M. A. **132**:119-121 (Sept. 21) 1946.

10. Ottoman, R. E., and Baker, J. W.: Metastatic Staphylococcal Infection of the Gallbladder, West. J. Surg. **53**:361-363 (Oct.) 1945.

11. Popper, H. L.: Pankreassaft in den Gallenwegen: Seine pathogenetische Bedentung für die Enstchung der akuten Pankreaserkrankungen, Arch. Klin. Chir. **175**:660, 1933.

12. Leon, W.: Acute Cholecystitis Following Unrelated Surgery, Am. Surgeon **20**:549-555 (May) 1954.

13. Schwegman, C. W., and De Muth, W. E., Jr.: Acute Cholecystitis Following Operation for Unrelated Disease, Surg. Gynec. & Obst. **97**:167-172 (Aug.) 1953.

14. Walters, W., and Snell, A. M.: Diseases of the Gallbladder and Bile Ducts, Philadelphia, W. B. Saunders Company, 1940.

15. Baker, J. W., and Koutsky, J. J.: Problem of Common Duct Exploration at Time of Cholecystectomy, West. J. Surg. **59**:495-503 (Oct.) 1951.

Plastic Surgical Principles in Farm, Industrial, and Traffic Accidents

Facial Injuries

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and
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Whatever is best for the patient is a proper surgical principle, without any specialized classification; hence, the subject, "Plastic Surgical Principles," may be open to some interpretation. Concentration of types of patients and study of them and of results of their treatment, however, may lead to establishment of procedures that will prove the most beneficial.

In the repair of all injuries, as of those considered here, the basic surgical need is to do the reverse of what the accident did. This essentially means sorting out, cleaning, and putting back together displaced, torn, and avulsed soft tissue and displaced and broken bone, with preservation of all possible viable parts.

If severe facial injuries can be repaired soon after the accident, the best results are obtained and less secondary correction will be necessary. If, however, there is too much general shock, too much brain damage, a fractured cervical vertebra with possible cord damage, or intoxication, operation is delayed.

In severe soft-tissue tearing of the face, neither of two recommendations from World War I and World War II is applied. One such recommendation was to do a cutting débridement; the other was to pack the wound open. Neither of these need apply to the face (or to the hand), because gas gangrene does not develop in the face—the possibility of such development was the inciting factor in the promulgation of these two rules. Of great importance is the fact that more function and more features can be preserved by saving tissue.

With early operation, by patient sorting out and replacement of the torn tissues, and by saving every possible millimeter, the parts may be totally restored in a single operation. If either a cutting débridement or a packing open of the wound is done in severe injuries, necessary tissue may be lost; then a great number of operations would not restore the patient to as nearly normal a condition as would one early tissue-saving and -replacing operation. The tissues are débrided by cleaning out foreign bodies, dirt, and oil and removing detached or dead fragments, but nothing that might live is cut away from the face. (These same rules, of not cutting away tissue that might live and not packing the face open, also apply to the hand.)

In putting together a severely torn face, the various flaps and parts are identified and returned to their normal positions. Closure is started at known points—the border of the nose, the brow, the tarsal, or the lip border. These points are closed with deep 000 nonabsorbable surgical (silk) sutures if possible; if the flaps are too thin, with

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In presentation at the Western Surgical Association, plastic surgical principles of repairs in other parts of the body were summarized. Included were farm injuries involving the genitalia with total loss of skin and repair, burns, and resurfacing of the extremities. For publication these elements require too much space for discussion, and this first paper is limited to surgery of the face.

surface sutures close to the edges. It is important to avoid putting in wide surface sutures, because if they are put in far from the wound margins wide cross-wound scars will result. Such wide scars are called "ladder scars," and they cannot be eliminated satisfactorily, since there may be no way of getting rid of them without excising too much tissue to allow for smooth closure.

One type of injury crashes open the face, actually chisels the eyes apart, and makes an opening back to the ethmoid region, with displacement of the canthi. The correct position of a canthus that is badly displaced probably cannot be restored. A correct canthal level may be obtained, but the proper depth is unattainable if there has been severe distortion and long fixation. The internal canthal ligaments are attached by several dozen tiny filaments to the crista lacrimalis, and even the smallest sutures will not replace them very well. The best criterion is to bring the canthi back to where they came from, if possible, before fixation of the fibrous tissue and infection have occurred. This is usually a procedure of elevation and molding together of the separated areas.

Adequate anesthesia is necessary for these operations to be done satisfactorily, and intratracheal anesthesia, or at least an intratracheal airway with intravenous anesthesia, is usually relied on. In some institutions effort is made to close these wounds without any anesthesia, but in most instances such a procedure is not satisfactory. Even children may be operated on under local anesthesia soon after the accident. The wound is usually packed with a topical anesthetic, and then needles for deeper infiltration or block are introduced through the wound so that pain is reduced to a minimum.

Fractures of the facial bones at times are so extensive that there may be a great number of fragments with many little chips of bone, as though there had been an explosion. This results in a totally displaced and fractured face, so that the face may be one single plane from ear to ear, with the nose crumpled in, both orbits down, dental arch

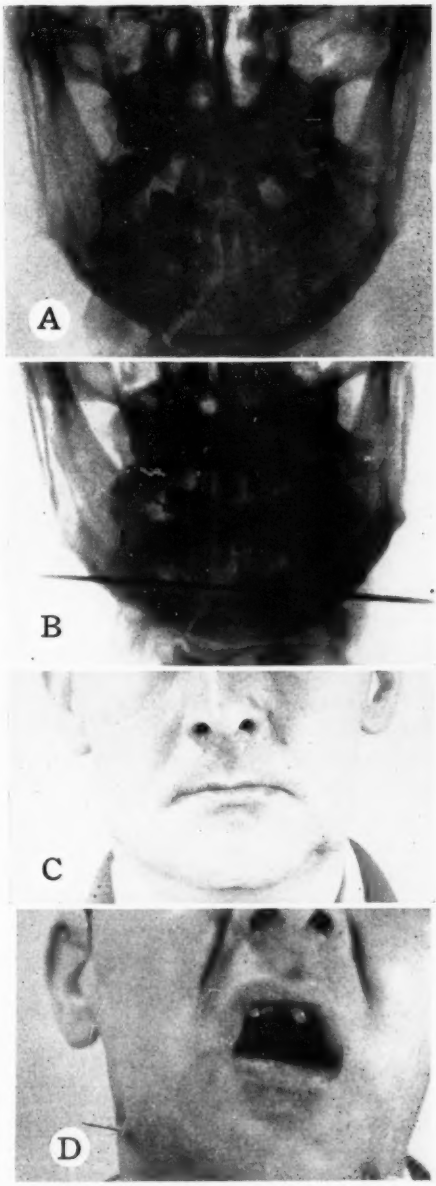

Fig. 1.—*A*, comminuted fracture of mandible at symphysis with inadequate teeth for satisfactory interdental wiring. *B*, *C*, and *D*, taken after reduction jaw stabilized by an internal "wire pin" driven across the fracture site. Wire arch placed around available teeth gives fixation in another plane. *C* and *D* show wire pin in place, fixing fragments but allowing for good opening and closing of jaw. Moderate chewing is possible during this stage of healing.

loose, palate split, pyramidal fractures on both sides, septum crumpled, and the lower jaw broken.

Basically it is necessary to do the opposite of what the accident did—pull the nose up out of the face, get the canthi into position, establish the airways, bring the teeth together so that the jaws close and occlude. The condition of the motor and sensory nerves and of the seventh and second nerves should be recorded before operation. Cervical-spine films should be taken in all cases of snap blows about the head. Any needed consultation, such as neurological, ophthalmological, otolaryngological, or dental, is obtained.

Functions of the face are protection of the eyes with the lids, provision of open airways for breathing, and to hold the teeth together so the patient can chew. A fourth function of the face is to appear normal so that the patient may have a chance at a normal social, educational, economic, and marital life. Therefore, in addition to the three basic physiologic approaches to the problem, the fourth problem is to try to get the face to look as it should, i. e., to reproduce the normal "contour anatomy," a term we have used to describe the appearance of normal surface structures.

In a severe crush with little if any support left, the nose, orbits, and whole middle third of the face may be simply "mulched" forward. This term is used for want of a better one and indicates that the multiple fragments are saved and carried into place and held without loss of structure, the airway is aligned and reestablished as well as possible. The nose is pulled up out of the face, the upper jaw is carried toward the midline, and the front of the face is actually "mulched" forward, with packing inside the antra to hold it in place. There would be no place to put an overhead splint with a very shattered face, the parts wholly detached and displaced, it being something like an egg that has been dropped on the floor. The nose is held up after elevation by two wires from side to side through the fracture lines.

An important thing for consideration is the relation of bone and soft-tissue injuries. One patient may have a totally shattered face as far as bone is concerned with very little soft-tissue injury, whereas another patient may have his face totally disrupted from soft-tissue injury but have no bone injury. But injuries of both bone and soft tissue very often go together; for this reason it is thought that one should not follow a rule to wait 10 days before repairing facial injuries. It is believed that early repair should be done soon after the injury, if possible, and the advantages of early operation can be vouched for by practically every house officer who has observed patients under both early and late treatment.

Internal wire fixation may be used for fractures of both the lower and the upper jaws. For zygomatic fracture and orbital displacement one or more internal wires are placed across the middle third of the face from the sound zygoma on through to hold the fractured left side. The wire pin acts as a cantilever support, having a solid fixation on the solid side and coming through the face and picking up the fractured side after it has been replaced. If correct replacement cannot be done, of course there is no reason for putting the wire in place.

Diplopia may occur if the orbital border goes back or down into the antrum, allowing the globe and orbital fat to follow. Good restoration of the comparative heights of the orbits may be obtained by placing a wire across from the solid portion on one side through the replaced loose portion after it is correctly elevated, either with a bone hook through the face or with an elevator in the antrum through the buccal fornix.

The final result may be achieved in a single operation, with the four elements of function restored: (1) closure of the lid to protect the eye, (2) dental occlusion for chewing, (3) patency of nasal passages for breathing, and (4) restoration of normal contour anatomy of the face.

In most traffic accidents the tissues are usually present but just displaced, but in some accidents, such as gunshot injuries,

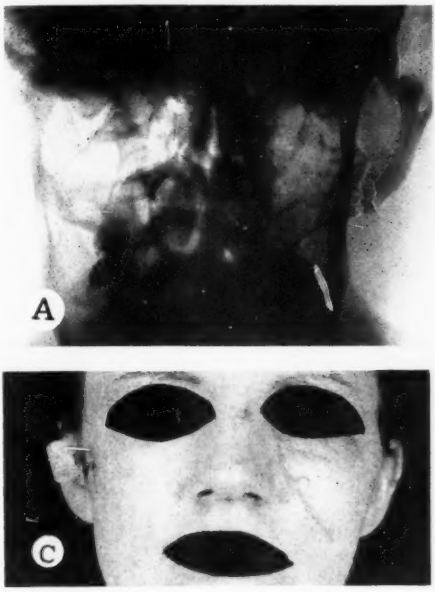

Fig. 2.—Fracture dislocation of the zygoma. *A*, some of the fracture lines have been etched in for clearer reproduction. Tearing of soft parts and fragmentation of zygoma in addition to dislocation from all of its attachments resulting from traffic accident. *B* and *C*, fracture dislocation has been reduced by external approach with large jaw hook and internally through fracture lines into the antrum. Solid fixation by "wire pins" driven across the face, using the opposite zygoma for cantilever support, are utilized until solid union results.

there is the added difficulty that some of the tissues have been lost or blown away.

A free graft, which we have called the composite graft, from the ear is one of the most useful forms of tissue replacement on the face. Total replacement of the ala of the nose is possible by a free graft from the ear consisting of two surfaces of skin and the intervening cartilage. The repair is done in a one-stage procedure. This procedure in the replacement of lost tissue gives much better and more normal-looking nostrils than heavy flaps brought in from a distance.

In any free graft it is necessary to get back to an adequate minute blood supply when opening the scar. If this is not done, a free graft will not grow. There can be free blood over the wound, but the tissues must be opened and the scar removed down into an adequate minute blood supply. A pattern of the nasal defect is taken from the ear with two surfaces of skin and intervening cartilage. The web at the crus of the helix is used if possible; otherwise a suitable area is selected. The ear is either repaired directly or slipped under a scalp flap and brought out later, repairing the ear defect with tissue that was behind the ear.

The seventh nerve is of deep concern for these patients in relation to the normal expression of emotions, to normal chewing, and to a normal socioeconomic life, which,

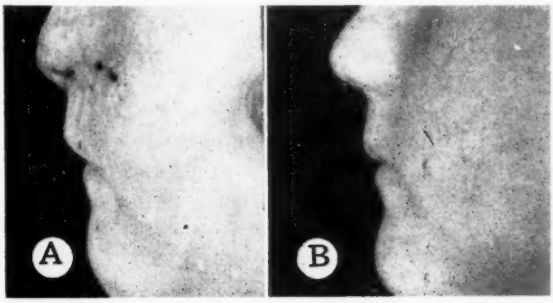

Fig. 3.—*A*, full-thickness loss of alar border and scarring of cheek, repaired in one operation with composite graft. *B*, addition to deficient nostril of a composite graft from the ear includes lining, cartilaginous support, and skin.

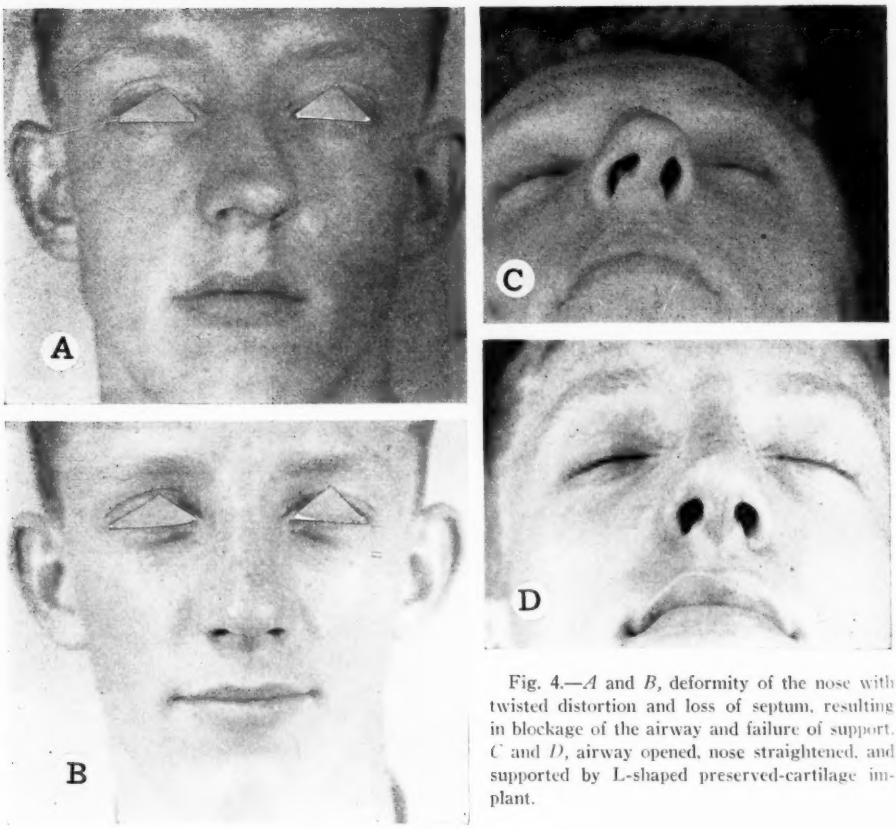

Fig. 4.—*A* and *B*, deformity of the nose with twisted distortion and loss of septum, resulting in blockage of the airway and failure of support. *C* and *D*, airway opened, nose straightened, and supported by L-shaped preserved-cartilage implant.

in turn, is dependent on normal contour anatomy. All patients with facial paralysis are passed on either by a nose and throat service or by a neurological service to determine whether either service does or does not wish to do something with the nerve. When no work on the nerve is indicated, fascia lata from the leg is taken and support is given to the paralyzed face with the fascia by transferring motion and support to the temporal muscle, which is supplied by the fifth nerve.

Since our first publication, in 1938, we have felt that this is the most satisfactory way of doing the substitution. There seems to be no reason for using metal wires or other foreign material when fascia lata is so easily accessible.

A patient may develop a nasolabial fold by a slight setting of the temporal muscle and show good emotional expression, using this fifth-nerve temporal muscle for the seventh-nerve facial muscle. It is important to have these patients appear somewhat glum on the unaffected side, and they are cautioned not to overact.

Even if nerve work is going to be done, the face can be supported while the nerve is regenerating.

In the consideration of late correction of a nasal deformity, it is important to know whether the deformity has occurred from disease or from a childhood injury. If a person with practically no nasal bridge has not had a septal abscess or had any bone taken out, one may find that bone still

present in the face. By going down into the face, what is left of the bone may be loosened along the frontal processes of the maxilla and may be brought up out of the face. This necessitates refracturing the bone with chisels or saws and recreating the first fracture; this late process is still reversing what was done in the accident.

When enough support for the nose cannot be found, it is important to decide what can be substituted for the support. Many substances have been used, but theoretically the best thing is the patient's own bone or cartilage. However, autogenous fresh cartilage is likely to distort badly, and the repair may have to be done again. Often the repair work has been well done, but the cartilage seems to curl too much in many instances. This difficulty might be avoided by using bone. Preserved cartilage may not last forever. It may break in two, but after the nose is once lined up it is not too much trouble to change the support if it becomes necessary. Bone transplants are not free from the danger of absorption, and many patients do not want to go through the second procedure of obtaining the bone graft, but the bone grafts at least do not curl.

Cartilage from animals has not been observed and recorded in a sufficient number of successful instances to warrant dependence on it at present.

Artificial subcutaneous prostheses for the nose, such as ones made of acrylic resin, have not been observed over a sufficient number of years to permit conclusions as to their value, but a prosthesis which proved permanently satisfactory would be one of the greatest helps to this type of reconstructive surgery. Experimental work is being done with silicones and polyvinyl compounds and gold at present.

The septum seems to be an important part of the nose, despite the teaching that it is unimportant as a support and the apparent lack of concern that is occasionally seen in resections of it. It seems evident that a normal nose cannot be pushed flat down on the face because the septum is there. Some have said that the septum does not make any difference in the lower part of the nose, and perhaps it does not if one considers syphilitic deformities in which there has been a loss of lining and the nose has been pulled up. And it may not make much difference if one has a heavy alar cartilage. However, many patients have been seen whose septums have been resected and who now have dorsal defects such that the tips of their noses could be flattened against their faces. It is very rare to see a patient who is not disturbed with a nose the tip of which one could push down on the face. So in these instances, where the deviated septum has been removed and a deformed nose has resulted, it is believed that the depressed dorsum should be supported. It can be supported with any subcutaneous prosthesis one chooses to use.

Nasal deformity often results from an early childhood fracture. Almost all children had falls; almost all of them have broken their noses, whether they know it or not, and in perhaps some of them the cartilage is torn loose, so that the injury may come very early in life and not show up until around puberty. It is possible that injuries may occur during delivery. There seems to be no reason for not correcting the result of such damage of the nose at a later date, whenever the patient wants it done. If the patient had had a normal nose at this late age and had had it laid over under one eye, he would come in and have it put back where it belongs; therefore there can be no reason for not bringing it back up where it belongs, even though the injury occurred early in life.

In gunshot and other blast injuries extensive loss of tissue always occurs, and so this type of facial injury always presents a complicated surgical problem, such as wide losses of the jaw from angle to angle and of the overlying soft tissues of the lower third of the face. The plastic surgeon has to build backward, in contrast to the usual concept of construction. In a building or almost anything else one would put the support in first and then put the outside walls

on it, but the surgeon has to reconstruct the outside first and then put in support, somewhat like erecting a tent rather than a solid building. For such losses a flap is brought up from below, and a bone graft of the body of the jaw is put in after the flap is in place. The bone graft from the full thickness of a rib or from the ilium is put into the covering flap secondarily. The patient is thereby given a watertight mouth, the saliva going down instead of pouring out.

There are several important points about overhead traction of the jaw. The fractured upper jaw does not always hang down. If the blow comes from below, such as when the upper teeth strike the steering wheel, the antrum, which is the basic support, crumples upward and in these instances the upper jaw needs to be pulled down in place, if it has impacted upward, and should not be pulled upward to a further degree. Elevation of the upper jaw most of the time can be obtained in about 30 minutes with enough elastic traction from the vertex to the chin. If the jaw will not come up, probably a piece of bone is blocking it or the fragments are fixed with scar. Of course, it may be too late to get the upper jaw up. Teeth are put together and the upper jaw is supported, using the lower dental arch against the upper with the chin-to-vertex elastic bandage. This is a simple method, but it is effective in many instances.

Overhead traction on a patient with a middle-third separation may pull the middle-third of the face too far up so that the walls of the antrum pass by themselves. The result after several months may be a permanently short middle third of the face, and the patient may have to have an extra set of teeth to wear over his own set. In such a case the middle third of his face has been so shortened by the excess overhead traction and has become so tightly fixed that it is a permanent deformity.

It is not recommended that one try to break up one of these faces that has had a great many fractures after it has been fixed for several months. In these patients seen long after the accident, it is not uniformly possible to take a chisel, reproduce the fracture, and get the face back where it should be; at least this has been our observation. Some readjustments and improvements might be made, but results cannot be obtained comparable with those that can be produced by early operation before permanent fixation has occurred. Repair in these instances is usually based on subcutaneous implantation of cartilage, bone, or other prostheses to build out the contour anatomy.

Burns occasion a great deal of discussion on all surgical services. In World War II surgery centers, one-third of the work was taking care of facial burns and restoring lost skin so that features could be put where

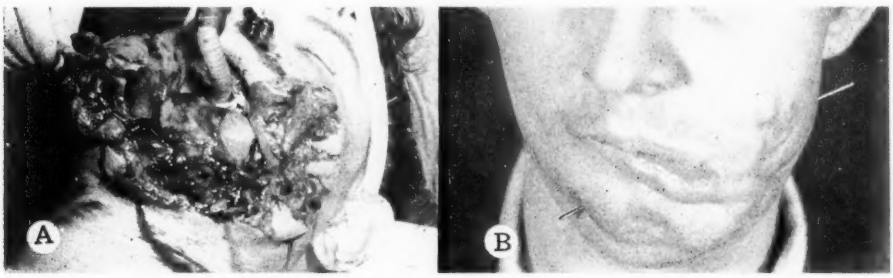

Fig. 5.—*A*, compound facial injury with extensive soft-tissue tearing and loss of bone resulting from gunshot injury at close range. Immediate repair done, closing soft-tissue defect as well as possible. *B*, soft-tissue collapse was prevented and the remnant of mandible stabilized by internal "wire-pin" driven across bony defect. Wire-pin still in place; later it was removed and a bone graft done.

they belonged and normal function and normal contour anatomy could result. Unfortunately, the statement has been made that it does not make much difference what is done for a burned face because all burned faces heal up. This is usually true, but if the full thickness of the skin is burned through, there will be scarring and distortion so that the four functions of the face will be lost. Therefore, the problem, from a plastic surgical standpoint, is the deep burn of the face, not the superficial burn, which will heal as well as superficial burns elsewhere on the body.

With loss and distortion of the eyelids, cheeks, nose, mouth, and neck that occur in facial burns, the problem still is to do the opposite of what the burn did. Take out the scar. Restore the tissues as close as possible to their normal positions and then graft in what skin has been lost.

Free-skin grafts are used wherever possible in facial burns, instead of flaps, unless a whole feature, such as the nose, has to be replaced. Free grafts allow the best contour and most natural surface and allow for some emotional expression; whereas, a flap is bulky, gets fat on any increase in weight, and does not allow freedom to express emotion.

SUMMARY

This paper itself is a summary of considerations and procedures following general rules of surgery most applicable to the face. There are some important individual considerations for procedures in this region, as outlined in the first and second paragraphs.

Burns in Children

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The subject of thermal injury has held considerable interest for researcher and clinician alike, as judged from the volumes of literature on all phases of the subject. Most studies dealing with treatment are of a general nature or discuss therapy with reference to all age groups. There are many articles concerning burns in children by British writers, such as Colebrook [6] and Jackson.[11] In general, the emphasis seems toward adults, which is paradoxic, since the majority of burn patients are children.

Increasing concern is now being shown about the incidence of burns in children. This is logical, for it parallels the relative and absolute increased importance of trauma as the greatest cause of infant and childhood mortality and morbidity. Of the forms of trauma burning is the single commonest injury. One by one the infectious diseases of childhood are being brought under control, being modified, or being prevented by the antibiotics and biological products. What were once the scourges of children are now being relegated to the background, and, consequently, the poorly controlled killer of children, trauma, emerges prominently.

These changes have not transpired unnoticed. Dietrich [8] forcefully posed the problem of childhood accidents as a public health responsibility and referred to their prevention as a vaccine which should be administered by the medical profession as other types of vaccine. He emphasized the increasing importance of trauma in child mortality. This contention is supported by others, also. Colebrook and Colebrook,[6] in speaking of burns specifically, have even suggested the use of mechanical devices to protect children from contacting heating appliances. It has been proposed that clothing manufacturers change their emphasis from long-wearing and washable garments to fire- and heat-resistant fabrics for children. In the summary of an article on the treatment of burns in children by Kiskadden and McDowell [12] appears a statement that "burns are a preventable accident." This concept keynotes the feeling of most writers on the subject.

Those who work in centers or on wards where many burned children are treated eventually develop the philosophy that all burns are preventable exclusive of some unusual type of disaster involving many people or families. It is hard to fix the blame in all cases, but proof of responsibility hardly compensates for the loss of a child's life. The inexperienced, naive child explores and tests, often getting burned literally as well as figuratively. Careless adults allow unprotected hot objects and liquids to be available to the curious child. The advertising slogan "tender as a baby's skin" is well applied in its literal sense, for what often seems to be a completely benign form of heat to the calloused hands of the parents may have a devastating effect on the child.

As in the case of the infectious diseases, there is no single line of attack that will bring about the control or prevention of burning in children. There are numerous etiologic factors, and the elimination of one does not solve the problem. The causes and distribution seem to fall into certain patterns, however.

The factors of age, thin skin, and "modus operandi" of burns in children lead to certain differences in the type and surface

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distribution of burns when compared with those of adults. Scalds in adults are generally small in area and shallow in depth. The youthful epidermis, however, does not withstand hot liquids nearly as well as the adult skin, and many full-thickness wounds result. These burns are common on the scalp, face, neck, shoulders, and upper trunk as a result of overturning hot liquids from the stove or dinner table. We have observed some severe burns of the face, neck, and shoulders from spilling of hot grits. The scalp is an infrequent site for burns in the adult, and burns are seen there only as a result of bizarre industrial accidents. Scalds of the feet and buttocks are commonplace in children who are often bathed in makeshift tubs with poor control of the temperature of the water, which is supplied from some vessel heated on the stove. The child either stands or is seated in the water before it is realized that the heat is too great. Thermal accidents involving the feet occur from other sources also owing to the fact that small children frequently do not wear shoes. When the children reach the age at which they are able to be outside, away from the watchful eye of mother, they become vulnerable to flame burns when their clothing becomes ignited in trash fires and the like. Flame injuries are in most instances severe full-thickness wounds. The little boys suffer wounds of the lower extremities from burning trousers, and the girls acquire encircling burns of the trunk when their dresses become ignited.

Unlike adults who suffer burns of the dorsum of the hand, children suffer burns of the hand that usually involve the palm from grasping hot objects. Wary adults are not likely to make that error. Mangle burns of the hands are more likely to be suffered by curious little children than by adults and usually involve the dorsal surface of the hand. The mangle burns constitute burn plus crushing injury, and, in addition to the skin loss, there is often damage to the tendons and nerves and to deep muscle if the forearm is involved. As a rule, contact burns are small in area and rarely serious. The exception is the infant, whose tender skin can be damaged seriously by direct contact with hot water bottles which are placed next to the skin to provide warmth. This is an extremely dangerous practice and is to be condemned.

Parents inadvertently may produce burns in their children when bathing them. This is particularly true when facilities require that the water be heated on a stove. A parent may place a child in a tub of heated water, forgetting that the metal container is dangerously hotter than the fluid it contains. It is also true that an adult's gross evaluation of the temperature of the water is much too inaccurate and the thin skin of the baby is scalded rather than cleansed. The purchase of a thermometer is a small price to pay for the prevention of this accident. Unfortunately, instances have been seen where the parents, angered at the child's soiling, hurriedly, and even at times maliciously, hurl the defenseless child into a tub of scalding water. This, of course, can lead to a burn of extensive surface involvement.

The steam inhalator, while undoubtedly a very effective therapeutic device, is also a dangerous one. Too often they are pulled over or fall, spilling the boiling contents onto the child. This is frequently a very serious accident, since the victims are usually rather young, and to the severe burn which follows is the added hazard of a preexisting and, presumably, severe upper respiratory infection. The treatment of shock and management of the local wound are very difficult in these cases.

From the foregoing it is evident that prevention of burns may be difficult but not impossible. Parents should be advised as to the dangers of heat to the sensitive skin of infants and children. Greater care should be exercised in the kitchen and bathroom to avoid accidents. Heating devices should be out of the reach of small children or provided with a protective screen. Steam inhalators can be anchored to prevent falling. Great care should be exercised in the use of lye about the household; not only may little

children drink it, but they may crawl into it when spattered on floors and sinks.

TREATMENT

As in the case of infectious diseases, prevention is ideal, but with trauma the main emphasis must still be on treatment.

There are special problems concerned with the treatment of burned children which will be discussed herewith. This report is designed to present a practical plan of management of burned children that can be applied to individual cases or instituted in a large center where many children are cared for.

As in adults burns in children produce open contaminated wounds of variable depth and are capable of causing shock. The concept of the thermal injury as an open wound is rigidly held, and the objective of treatment is to convert the open contaminated wound into a clean closed wound at the earliest date with the least amount of residual disability to the patient. Shock is energetically and aggressively managed. Finally, convalescent rehabilitation is regarded as an important adjunct to total care.

Hospitalization offers the most effective facility for handling the traumatized child. It is advisable to admit any burned child to the hospital, regardless of suspected burn depth, when the surface area involved is at least as large as the so-called "flash areas," i. e., face and hands. It must be borne in mind that the thin skin of the child may be damaged more seriously than experience with adults would lead one to suspect. Also, the ratio of surface area to blood volume is less than that of adults, and children may evidence shock with burns of far less than 25% surface area.[5] An adult, particularly in the older age group, may likewise evidence shock with areas less than 25%, but the tendency is not nearly as great. Generally the error committed even by experienced surgeons in evaluating the burn in children is to overestimate the extent and to underestimate the depth. For these and other reasons a conservative rule would be to hospitalize the child when there is doubt.

The general plan of hospital care is to institute treatment of the local wound and the systemic reaction immediately and simultaneously. It must be emphasized to all persons concerned in the management of the burned child that the area of burn is an open wound subject to infection and capable of producing shock and even death or other less severe complications, if not properly treated. Obviously, lifesaving takes seniority, but the aim to convert this burn wound into a clean closed wound must be constantly stressed.

Local Care.—On admission to the hospital, the patient is placed in a clean dressing room designed for burn care and where operating room rules of asepsis are enforced. Masking of all persons in the room is regarded as the best prophylaxis against the addition of infection.[3] It seems illogical to assume that the heat of the burn kills the skin bacteria, for many must survive even severe burns in sweat glands and hair follicles. At the same time, the necrotic and seriously damaged tissue is excellent culture medium for the organisms harbored in the human nose and throat. While preparations for wound care are begun, the systemic treatment is started by introducing a needle into a vein, cutting down, if necessary, drawing a blood specimen for baseline studies, and starting an infusion, usually of plasma or a plasma expander. If these are not readily available, however, some other isotonic solution is used to maintain the patency of the vein. Tetanus prophylaxis is given routinely.

The history obtained from the parents or other witnesses is helpful. An accurate description of the mechanism of the injury can be useful in estimating the probable depth of the burn. It is important to inquire about the presence of upper respiratory infections, nutritional problems or drug allergies. A record of the child's past history, recent exposure to infectious disease, and immunizations should also be ascertained from parents or pediatrician.

While these immediate steps are being taken, the surgeon or an assistant has scrubbed his hands and donned a sterile

gown and gloves. After sterile draping of the patient, the burned area is very gently washed with white soap and water, using soft squares of sterile cotton. This can be a remarkably painless procedure, providing the water is lukewarm and the washing is done deliberately, gently, and unhurriedly. Adults have been questioned regarding pain during this "prep" of their burn wounds, and most have said it was not at all painful, whereas a few described it as being only somewhat uncomfortable. The soapsuds are flushed away with copious amounts of sterile water or saline. Foreign bodies, bits of loose skin, and blebs are then very carefully removed with sterile instruments. The practice of leaving the blebs of devitalized epidermis in place to act as a protective covering is believed to be unwise. No attempt, however, is made to excise into bleeding tissue. The cleansing and so-called débridement of an acute burn requires a high degree of gentle surgical technique and is the key to proper wound preparation.

After cleansing, a compression dressing is applied. It is firmly believed that healing and prevention of contamination require sterile protection and that gentle and even pressure over the wound area aids in the prevention of stasis and the resorption of the edema that invariably accompanies trauma.[13] It has been shown repeatedly and is time-honored that healing and immobilization are closely allied, and it is more feasible to splint the part when it is first covered by a bulky, firmly applied dressing. We have not adopted the open-air treatment of burns. It has seemed to us to be highly impractical, particularly in children who cooperate poorly. The great amount of nursing care needed is another factor which, it seems to us, is against it.

Dressing of the wound is greatly facilitated by the use of a prefabricated flat sterile pad (burn dressing) that contains 24 layers of cellucotton covered by a single layer of fine-mesh gauze.[2] This dressing comes in several sizes, two of which are especially suitable for children. One is just right to cover the upper extremity, and the other is larger and will accommodate the lower extremity or the trunk. These dressings can be applied quickly to the area and serve as the only dressing needed. If desired, the burn may first be covered by a single layer of fine-meshed gauze lightly impregnated with petrolatum, followed by the compression dressing laid over it. The petrolatum gauze simply aids mechanically in the subsequent removal of the dressing and has no other value. Topically applied medicaments are not necessary, and their avoidance obviates the risk of sensitization. This resilient pad is placed over the petrolatum layer and bound firmly by circular turns of woven elastic bandages to provide even resiliency throughout. The wound is then splinted with plaster molds, padded boards, or shaped aluminum splints. The hands, if injured, are immobilized in the position of function. It is very important in dressing hands, to keep the fingers separated with gauze dressings to prevent the development of intertriginous areas of maceration. The toes should likewise be separated. The face is generally covered with light dressings, or, if pressure is applied, the bony parts are well padded to avoid undue pressure to the ears and eyes (cornea). Nostrils are always left open, and the eyes are usually not covered. Occasionally, it is impractical to apply dressings to the face, and the open technique is used. However, it is to be noted that more edema develops under open treatment than when pressure is used. Burns of the perineum can be covered with a sterile diaper or a layer of petrolatum gauze, and dressings should be changed frequently to minimize fecal and urinary contamination.

Opiates and anesthetics are not advocated in the initial wound care or in subsequent dressing changes. Beecher [4] has pointed out the dangers inherent in the use of morphine in traumatic and burn shock, and the risk of general anesthesia at this time is well known. It is seldom necessary to resort to the use of drugs for the relief of pain during initial care. Gentle handling of the patient in a warm environment and early coverage of the wound have proved effective in allay-

ing most of the discomfort. Occasionally, a suppository of one of the barbiturates is given to quell the fear that accompanies the admission of a child to hospital. Subsequent dressings, if done quickly and carefully, using the prefabricated dressing previously described, are quite painless, and drugs and anesthetics are not indicated. The use of these agents interferes with feeding, which is so extremely important in the total care.

Systemic Care. — Systemic treatment is started simultaneously with the care of the wound. Immediately on admission a needle is introduced into a vein, blood samples are withdrawn, and intravenous fluids are started. This is important, since, if shock should develop and venous collapse occur, it may be very difficult to start intravenous therapy. Formulae based upon weight, surface extent of the burn, or certain laboratory indices may be unreliable as rigid guides to treatment of burn shock in children. The shock picture changes from hour to hour, and treatment must be closely supervised on that basis. One cannot simply transcribe a stereotyped plan to the patient's order sheet. Close observation is a prime requisite. Clinical observation is of greater importance than the newer laboratory techniques and recent advances in the theory and practice of fluid and electrolyte therapy. The flame photometer will never replace the conscientious surgeon. The most reliable and simplest laboratory index is the accurate determination of the hourly urine output and specific gravity.

Whereas the hematocrit readings, size, weight, and blood volume of the patients vary tremendously, the normal urine output in children and infants is fairly constant. The normal amount is believed to be from 15 to 35 cc. an hour, with a specific gravity of about 1.016. An indwelling catheter should be inserted into the bladder on admission, and hourly urine specimens can be obtained and their quantities and specific gravity measured. This also permits the early detection of hemoglobin in the urine. It is thus easy to follow the patient's progress from hour to hour. The correlation of the urinary findings with the clinical appearance is an excellent way to evaluate therapy.[1]

Other laboratory determinations, such as cell pack, hemoglobin, and plasma chemical levels, are carried out. Isolated determinations are often misleading, but repeated tests will aid in indicating trends in the status of the patient. The laboratory is much more valuable later in assessing the nutritional status of the patient than it is during the shock phase.

Burn shock is due to reduction to greater or less extent of the circulating blood volume and is characterized clinically by excessive thirst, restlessness, hemoconcentration, oliguria or anuria, and progressive signs of peripheral collapse. Cyanosis, lividity, coma, and respiratory collapse are late and are evidence of irreversible shock, and therapy should be directed to the prevention of these terminal events.

Children and infants cannot be regarded as adults in miniature with respect to burn shock. The ratio of blood volume to the skin surface area is smaller in the child than in the adult, and the difference is still greater in infants and the newborn. The reaction to thermal injury is dramatic in its rapidity of onset and violence. The chain of events leading to terminal manifestations may occur with frightening rapidity and with seemingly little stabilizing natural response unless early and energetic treatment is carried out. Small infants and the newborn can acquire the appearance of a terminal patient within a few hours. The loss of fluid from the burned surface and into the surrounding tissues as edema can quickly reduce the circulating blood volume and intracellular fluids to a lethal level. Coma and convulsions can occur very early in burn shock in these little patients.

The thirst associated with burn shock may be extreme and is generally not relieved by oral intake of fluids, which more frequently leads to vomiting and further increase of fluid loss. Children and infants vomit more readily than adults in the presence of established shock. It is better to combat the reduced circulating blood volume

with its accompanying thirst by the use of plasma, whole blood, or one of the plasma expanders. As the circulation is improved by the parenteral therapy, generally within 10 to 12 hours, oral intake can be begun cautiously. Depending upon the size and weight of the patient, feeding formulae, milk, or water may be given, starting with 1 oz. an hour in the smaller children to 5 oz. in larger ones. It is not expected, however, that the intake by the oral route alone can reverse an established shock state.

Whole blood is ideal for the replacement of the circulating fluid volume in the treatment of burn shock. It provides not only volume expansion but also vital oxygen-carrying red cells to replace those damaged by the burn and hemoconcentration.[10] Hemoconcentration is not a contraindication to transfusion therapy. The hematocrit measures only the relative cell volume and does not reflect the physiology of the available cells. The life of the red cell is diminished after a burn, and the use of whole blood during the shock phase helps to diminish the inevitable anemia which follows large burns.[10] The blood can be given in a ratio of one part blood to two parts plasma or expander. This is not a rigid rule but is a practical guide and varies with the case. Oxygen should be used when respiratory distress is present or imminent. Burn shock usually lasts for from 40 to 48 hours, and it is during this period that frequent and careful clinical observation is necessary. If the urine output is about 20 cc. an hour with a normal specific gravity and the clinical signs of shock are absent, it can be assumed that treatment is adequate. Pulmonary edema due to overzealous fluid therapy is not common if judicious individualized parenteral therapy is given.

The management of shock with lung burns is more difficult, since the pulmonary injury leads to edema, thus requiring greater caution in the intravenous therapy. Burns of the face are prone to involve the respiratory system, and manifestation of such involvement must be watched for. There is the upper respiratory tract burn characterized by singeing of the nasal hairs, redness of the tongue and throat, and hoarseness. If the respiratory burn is severe, stridor may develop and demand tracheostomy. Recovery from lung injury is common if shock can be controlled. True or deep lung burns usually develop from inhaling steam or fumes. The respiratory epithelium is directly damaged, causing edema, rales, and severe respiratory distress, and the patient exhales a frothy fluid. Tracheostomy is of little value in the deep burns, and recovery is uncommon. In animal experiments it has been shown that an inhalation of actual flame will not produce temperatures high enough to burn the respiratory epithelium beyond the major bronchi. However, the inhalation of hot moist air will damage epithelium well beyond this point.[15] Where evidence supports the diagnosis of respiratory tract involvement, it is advisable to keep the fluid intake a little less than the calculated optimum to avert overdistension of the pulmonary circulation. Rales in the chest or wheezing usually indicates overhydration and calls for a decrease in the rate of administration of intravenous fluids.

The hematocrit determination can sometimes be helpful in following the trend of therapy, but the use of whole blood makes this test somewhat unreliable. In children the normal range is from 35% to 40%, being slightly lower than in adults.[5] Lower than normal values may indicate a preexisting nutritional deficiency. A reversal of the albumin-globulin ratio is an almost constant finding in burn shock and may appear sometimes within 12 hours. The exact mechanism is not well understood, but studies have shown it to be due to decrease in albumin and increase in gamma globulin.[16] It may take several weeks or months for the normal levels to become reconstituted. Serious electrolyte depletion usually does not occur when shock treatment is started early. The early institution of oral feedings may be helpful in this regard. In addition, the majority of the ions are not actually lost but are rearranged in the fluid compartments, and as recovery from shock takes place there

is a shift toward the normal.[14] With delay in the onset of treatment, potassium, chloride, and sodium levels may show a minor decrease, usually during the second 24-hour period.

As soon as response of the shock to therapy is evident, oral feedings of a high-protein, high-carbohydrate diet are started, employing nasogastric tube if necessary to supplement or entirely replace oral intake. High-fat diets, while theoretically advantageous, often lead to diarrhea. An interval feeding containing 1 cal. per cubic centimeter is made, using protein hydrosylate, milk, egg, and sugar. This can be given in addition to a regular diet or can be used to provide the entire daily caloric intake. No rules of caloric intake have been set up, but the aim is to give saturation amounts of feedings. Even when shock has been severe, it is frequently possible to institute oral alimentation within 12 hours. Early feeding makes it unnecessary to supply parenteral electrolytes. The intravenous electrolyte solutions we are accustomed to use during the treatment of shock are mainly dextrose and small amounts of isotonic saline. They are given mostly to keep the infusion open between transfusions, and significant quantities of electrolytes are generally not administered.

The large open wound that is capable of producing shock is also an open surface that allows the continual loss of blood elements and plasma proteins. Exaggerating this loss is the increased demand for these same elements in an effort to combat shock and heal the defect. Other factors, not clearly understood, tend to augment the anemia and hypoproteinemia. For these reasons the necessity of maintaining high nutritional levels is paramount. A high-protein, high-carbohydrate, high-caloric diet is routinely given early, in anticipation of the late effects. However, synthesis by the already overtaxed liver is slow and inadequate, and replacement therapy is mandatory. Whole blood transfusions given during shock and utilized judiciously throughout the whole period of care are necessary to maintain high normal values for hemoglobin, hematocrit, and plasma proteins. Vitamin C, greatly needed for wound healing, is prescribed in large doses, up to 1000 mg. daily. This rigorous attack on nutritional deficiency is started with the initial care and is continued until final healing occurs.

Wound Closure.—It is very difficult to predict the depth of a burn on initial inspection, except on the basis of cause. The earliest that an accurate estimate can be made is at the time of the first dressing change, and even then the estimate is frequently equivocal.

The wound is first inspected on about the fifth postburn day, unless suspicion of infection necessitates earlier examination. At this time, the outer dressing is carefully removed down to the layer of petrolatum. If this layer is dry and adherent without evidence of necrosis beneath, a predominantly second-degree burn is present. If there is doubt, it is wiser to leave adherent gauze in place and reevaluate the burn two or three days later. The hurried removal of the dressings at this time can tear away regenerating epithelium and convert a partial thickness loss to a frank third-degree defect. A whole-thickness burn appears either as a dry, hard, charred, anesthetic eschar depressed below the surrounding skin, or as frank necrosis with moisture and early sloughing.[9] Frequently, there are intermixed areas of both full- and partial-thickness burn. In such instances the petrolatum layer can be lifted away from the full-thickness spots and be left adherent to the partial-thickness areas. If the demarcation of third degree areas is not definite, it is best to reapply the pressure dressings and to reexamine the wound at two- or three-day intervals until delineation is evident.

When diagnosis of full-thickness burn is established, usually at the initial dressing, a decision must be made as to the method of removing the slough. Simple daily sterile, moist, compression dressings, using the previously described burn pack, is an effective but slow way to prepare the wound for closure by grafting. It requires 10 to 14 days or even longer, and the danger of contamina-

tion is enhanced and the loss of vital elements from the wound is prolonged. Since a burn is an open wound containing necrotic tissue, sound surgical judgment would require that this "parasite" be removed by surgical excision when feasible. This procedure is indicated in any full-thickness burn where the demarcation is definite and whenever the patient's general condition is good enough to withstand safely an operation under general anesthesia.[7] Persistent shock, which can last for from four to eight days, is a contraindication. Poor nutrition, an upper respiratory infection, or some other complication would make this method unsafe. Face and neck wounds sequestrate rapidly because of their excellent blood supply and can be prepared by the dressing method, and wounds in this location, therefore, are never excised.

Surgical Excision.—The excision can be done as soon as whole-thickness areas of skin loss are well delineated, which is usually between the 5th and 10th postburn days.[1] This should, of course, be carried out in the operating room under strict asepsis and with general anesthesia. The area about the wound is cleansed with sterile soapy solution, and after sterile draping the slough is carefully dissected away with a scalpel in the cleavage plane between the dead and living tissue. Usually the excision leaves a most irregularly shaped defect. Hemostasis is secured by compression to control capillary oozing, while larger vessels are ligated with fine (00000 or 000000) silk. Whole blood is transfused at this time when the loss is sufficient to require replacement. In the case of the forearms and hands the excision may be carried out in a bloodless field secured by a blood-pressure cuff. In this case immediate grafting may be carried out, since the compression dressing over the graft will control the oozing. In other areas, however, the presence of troublesome oozing may interfere with the adherence of grafts. In such cases the excised wound is dressed with a layer of dry fine-mesh gauze covered with a bulky compression dressing. Wounds of the extremities are splinted. The nutritional state is reevaluated postoperatively and corrected if need be, and two or three days later the patient is returned to the operating room for skin grafting. At this time, the wound is seen to be dry and healthy. In this short interval there are obviously no granulations, but, rather, one sees normal-appearing subcutaneous tissue.

Grafting.—If the daily dressing technique has been used to remove the necrotic tissue, grafting can be done as soon as dead tissue is no longer present. It is not necessary to await the appearance of velvety granulations. A red, healthy, flat wound that bleeds easily when the fine-mesh dressing is removed is ideal for the acceptance of grafts. An excised wound is ready to close when the patient can tolerate an anesthetic. In either case the nutrition must be good and there must be no complication that contraindicates a general anesthetic. Blood transfusion may be necessary at the time of operation, and provisions for it should be available.

The donor areas are prepared by cleansing with soap and water. The thighs and the back are the most suitable sites. In the small infant and the newborn the back is preferable, since it is relatively long and flat, whereas the extremities at this age are small, fat, and chubby, and in the infant there are deep folds which make the removal of grafts difficult. The grafts are simply and rapidly cut and applied to the wound as a skin dressing. Children and infants have such thin skin that great care must be used to avoid taking too deep a graft if a dermatome is used. We prefer freehand cutting. The skin is transferred directly from the donor areas to the wound. Wrapping of the grafts in saline-soaked gauze or submerging them in saline solution is avoided, since the saline removes serum which seems to help fix the graft to the surface. Leftover pieces of skin that have been removed in excess of the wound requirements are saved, placed in a sterile container, and put in the refrigerator at ordinary refrigerator temperature (not frozen) to be used later if necessary to replace grafts in the event there is a partial slough. Similarly, if for some reason the operation must be terminated before the grafts can be ap-

plied, they can be saved and applied later, any time up to three to five weeks after removal, in the same way as a dressing is applied. These "ice box" grafts take quite well.

Time-consuming suturing of grafts is not done. The grafts adhere very well without suturing, and the slightly improved cosmetic appearance does not justify the time consumed, especially with large grafts. The grafted wound is dressed with pressure and splintage, using an initial layer of petrolatum gauze. The donor site is dressed in like manner and left undisturbed for two weeks, at which time these areas are usually healed if the patient's nutrition is satisfactory. The entire grafting operation can be done rapidly with very light analgesia.

In extensive burns homografting may well be a lifesaving measure and may tide the small patient over a critical period. It must be regarded as only temporary, since the grafts must eventually be replaced with the patient's own skin. It might be better to look upon homologous skin simply as a very fine burn dressing which may be left on for long periods of time. This skin seldom lasts more than 30 days, and healing of these grafts never takes place. Removal of this skin and grafting of the area is by no means as easy as initial excision and autogenous grafting.

Subsequent Care.—The grafted wound is inspected on about the fifth postoperative day unless infection is suspected earlier. The dressing is gently removed down to and including the petrolatum layer. Care must be taken to avoid disrupting healing grafts. If the nutrition has been good, immobilization adequate, and infection absent, the takes are good. Even in the presence of low-grade superficial infection, partial takes are common. Sometimes the first postgraft dressing is disappointing. The pigmented outer layers of the graft may come away with the dressing, giving the impression that the entire graft has failed to take. Actually, a thin pearly, shiny layer of healthy epithelium may be present, and on subsequent dressing obvious epithelium can be detected. Gentleness in changing dressings should be observed so that this semitransparent layer is not destroyed.

In cases where donor skin has been insufficient to allow complete closure at the initial operation, donor sites must be reused. It requires from two to four weeks for a donor site area to regenerate sufficiently to permit a new graft to be removed, and during the interim the patient's condition must be watched to avoid nutritional deficiencies.

As the wounds heal, dressings are gradually reduced in bulk until all are eliminated. An attempt is made to discard circular dressings and splints as early as progress of healing permits. Dressings are changed just enough to keep the wound clean and adequately covered. Dressings that are adherent to a clean surface are not forced away. It must be remembered that, just as the thin epidermis of children is vulnerable to thermal trauma, the new-growing epithelium is subject to injury by too frequent or roughly executed dressing changes. Any crusts and scales that are present on freshly healed wounds can be washed gently with soap and water. The application of hydrous wool fat (Lanolin) later on to grafted areas helps to make the skin softer and more pliable. Action motion and early ambulation or physical therapy are started as soon as sound healing permits.

THE OLD BURN

The ideal of early initial treatment with continuity of care until final rehabilitation does not always prevail. Many cases come to the hospital 48 hours to many months after the onset of the burn. During the interval, they may have had inadequate care at home or in circumstances where facilities have been limited. It is from these cases that one learns the extreme importance of early wound closure. If the wounds are large and the delay in closure has been long, these patients show marked nutritional deficiencies. They are often pitiful sights. They are frightened, pale, and emaciated, with foul-

smelling open wounds, either frankly infected or covered with pale exuberant granulations. Joint contractures and decubitus ulcers are often seen. The protein content of the plasma is often below the critical oncotic level, and there is hyperglobulinemia. The anemia is usually very pronounced, and large amounts of blood are required to correct it. Children display a pronounced weight loss that begins within a few days after the burn and proceeds rapidly to severe emaciation if the wounds are not closed. This marked weight loss is a cause for concern in these neglected burns.

Whereas wound infections are rare in the cases seen early and treated aggressively, these cases seen late nearly always have infections to a variable degree with a copious, foul-smelling wound discharge. The patients have a persistent fever and eat very poorly. Many have become dependent upon opiates for sedation. In these cases, also, there is seen the feeble attempts of healing by scar-tissue epithelium, which is thin and inelastic and produces great deformity. One becomes impressed with the need to prevent this state.

These cases can be considered nutritional emergencies, and without further delay the state is corrected rapidly by replacement therapy. Blood is transfused in amounts to achieve high normal levels of protein and hemoglobin. It may be necessary to institute tube feedings to give the patients adequate amounts of protein and to insure an intake of 3000 Cal. or more daily. Concomitantly, the wound is prepared for grafting by daily or twice daily dressings to remove infected and necrotic tissue. Piled-up and pale granulations may be cut away, or often they become flat and healthy under the influence of compression. Grafting is planned for the earliest date when the wound and the general condition allow. By intensive treatment this can usually be accomplished within a week. In one such case grafting was done on the third hospital day. Wound closure and subsequent treatment are the same as for other cases.

COMPLICATIONS

The acute contagious disease of childhood constitute the most important complicating factor. Many excisions and graftings are delayed because of isolation for one of these infections. Some of the burned victims actually acquire one of the diseases in the hospital and must be transferred to the contagious disease section. During the late winter and early spring months, particularly, hospitalization is prolonged in a high percentage of the cases and the end-results are sometimes less satisfactory. Upper respiratory infections are also prevalent, and, in addition to providing a morbidity hazard, they, too, interfere with early wound closure because of the risk of anesthesia in such patients.

Children are, of course, subject to the usual anesthetic risks as are adults. These have not been severe in our experience, and, although bronchopneumonia has occurred, there have been only rare cases of atelectasis. No proved cases of pulmonary embolus have occurred in our patients.

Many patients with severe flame burns with large areas of full-thickness loss show a hemoglobinuria, which occurs within a few hours after admission to the hospital. By far the majority of these recover without evidence of residual kidney damage. However, lower nephron nephrosis may complicate the recovery in some cases. Those cases that have not survived this complication have been those with extensive burns and with such profound shock that eventual recovery seemed unlikely, even without this added difficulty. One must always rule out the possibility of transfusion reaction as the cause of hemoglobin in the urine.

Infected wounds are rare in the cases that are treated soon after the onset of trauma. The burn wound is an open, freely draining surface, and as long as retentive agents, such as escharotics, are not applied there is little opportunity for the growth and invasion of surface contaminants. Sterile precautions throughout care minimize contamination. Early excision and closure of the

wounds preclude the possibility of organisms multiplying in the necrotic tissue. Antibiotics are not used prophylactically in all cases but, rather, are reserved for the extensive burns and when specific indications arise. Penicillin is the most commonly used drug.

Contractures at flexion creases are inevitable in full-thickness burns, but they can be minimized if the wounds are closed early and if grafts are placed in such a way as to produce scars parallel to the crease. Early motion helps to keep joints mobile and prevent fixation of contractures. When the final contraction of the scar takes place, usually several months after healing, such contractures and scars as interfere with motion can be corrected by appropriate plastic procedures.

Weight loss, anemia, and liver function changes are always present to some degree in severe burns and are thus not considered as true complications.

SUMMARY AND CONCLUSIONS

Burns are usually preventable childhood accidents.

A plan of management of the burned child is presented, with emphasis on the fact that the burn is an open contaminated wound that should be converted into a clean closed wound, with use of surgical excision when feasible to hasten removal of necrotic tissue to produce a surface that can be closed by skin grafts.

Shock is treated aggressively, using blood and plasma or plasma expanders to restore the decreased circulating blood volume. Careful clinical observation and the measurement of the hourly urine output are the principal guides used to evaluate the progress of the treatment.

The importance of the nutritional state is emphasized. Whole blood is used to replace elements lost through the open wound. High-caloric, high-protein, high-carbohydrate diets are used, with supplementary tube feedings if sufficient amounts are not taken by mouth.

DISCUSSION

Dr. Michael L. Mason, Chicago: Those of you who knew Harvey Allen, know how terribly seriously he took this problem of burns, not only in children but in adults. His philosophy of care of burns has been very nicely expressed by Dr. Day, that is, that a burn is an open injury and should be treated in the same way we treat any such injury—by cleansing, by excising, and by closing.

Of course the handling of burns must be done in stages and cannot be done as we would treat an open abrasion or other types of open injury, by a single primary operation. It was his feeling (and I am sure it is the feeling of many of us) that this early, very aggressive care, not only for the local wound but for the general condition of the patient, can obviate many of the serious complications that we see in burns.

The development of a serious electrolyte and protein imbalance can often be prevented or certainly can be minimized by early attention to the administration of blood and other proper fluids, in the same way as the care of the open injury can be simplified by an aggressive attention to the local wound.

Dr. Henry N. Harkins, Seattle: I should like to ask Dr. Day if the grafts are applied immediately upon excising the third-degree area or whether he sometimes waits 24 to 48 hours after débridement before applying the grafts.

Dr. Paul W. Greeley, Chicago: First of all, I should like to congratulate Dr. Day on the very fine presentation he has made this morning.

There was one phase of his paper, however, that was not mentioned, which I do believe should be brought out at this time. We are living in a machine age. One can buy equipment that will cut a skin graft just as easily as one can cut one's lawn. However, the machine which you buy to cut your grafts does not do your thinking for you. One has to do considerable long-range calculation as to how one is going to cover some of these very extensive burned areas.

Certainly faces and hands should be given priority. Large joint surfaces should come next, especially those of the neck, axilla, popliteal spaces, anticubital spaces, and so on. One should keep in mind how one is going to use the available donor sites. Many times it is impractical to try to accomplish what would be an ideal reconstruction at the time of the initial coverage just because of the magnitude of the burn and contamination of the wound. Therefore, it is imperative that we cover the important areas first with good pieces of skin, and where you fill in between, if you use little pieces, it does not make too much difference provided you get adequate healing.

It is very important, however, that we save and preserve large available donor sites of skin that can be used for later reconstructions. There is nothing more discouraging than to inherit a severely contracted burn involving the neck, axillae, elbows, and hands and find that a little piece of skin has been taken from the abdomen, another from the right lower quadrant, a little from the thigh and a little from somewhere else, so that we are left with no large piece of skin with which to make a good coverage for an extensive surface defect. If long-range early calculation is done, much more can be accomplished for these patients.

DR. SHERMAN W. DAY, Seattle: I should like to thank the discussers for bringing out very important points that I was unable to cover because of time.

I should have mentioned in the paper the point that Dr. Harkins asked about. We do not necessarily cover the excised area at the time of the original operation. This is done only when a bloodless field can be provided by the use of a blood-pressure cuff. In other words, when the burn is on a distal extremity, then we generally cover the area at the time of the excision. Also, areas are not covered at the time of excision if the burn is extensive. This prolongs the operation, and little patients do not tolerate surgery as well as do adults; at least this has been our experience.

Generally speaking, a delay of 24 to 48 hours is permitted between procedures, at which time the patient's nutrition is reestablished, the blood is replaced if any has been lost, and the patients are returned to the operating room and the grafting procedure carried out rapidly.

Anesthesia is used only long enough to obtain the skin, which is usually taken by a freehand knife. The anesthesia is discontinued while the grafts are merely laid on the wound, in much the same way as you would apply a dressing. This saves anesthesia, and I belive it also saves the patients.

I want to thank Dr. Greeley for his comments regarding the late convalescence of these patients. He brought out some very important points which I appreciate his covering.

Covering these large, open defects certainly is a problem. One way we get around it in some cases is to reuse original donor sites. Children's wounds heal rapidly if their nutrition is good, and we have been able to reuse the same donor site sometimes within two weeks.

REFERENCES

1. Allen, H. S.: The Treatment of the Burned Wound on the Experience of 1000 Cases, Ann. Surg. **134**:566, 1951.

2. Allen, H. S., and Bell, J. L.: An Efficient Dressing for Burns or Other Large Wounds, Quart. Bull. Northwestern Univ. M. School **25**:1, 1951.

3. Allen, H. S., and Koch, S. L.: The Treatment of Patients with Severe Burns, Surg. Gynec. & Obst. **74**:914, 1942.

4. Beecher, H. K.: Resuscitation and Anesthesia for Wounded Men: The Management of Traumatic Shock, Springfield, Ill., Charles C Thomas, Publisher, 1949.

5. Beil, J. L.; Day, S. W., and Allen, H. S.: The Management of Burn Shock in Children at Cook County Hospital, Quart. Bull. Northwestern Univ. M. School **27**:14, 1953.

6. Colebrook, L., and Colebrook, V.: A Suggested National Plan to Reduce Burning Accidents, Based on a Study of 2000 Cases, Lancet **2**:579, 1951.

7. Cope, O.; Langohr, J. L.; Moore, F. D., and Webster, R. C., Jr.: Expeditious Care of Full-Thickness Burn Wounds by Excision and Grafting, Ann. Surg. **125**:1, 1947.

8. Dietrich, H. F.: Prevention of Childhood Accidents: What Are We Waiting For?, J. A. M. A. **156**:929, 1954.

9. Harkins, H. N.: The Treatment of Burns, Springfield, Ill., Charles C Thomas, Publisher, 1942.

10. James, G. W., III, in Symposium on Burns, National Academy of Sciences, National Research Council, Washington, D. C., 1951.

11. Jackson, D. M.: Burns and Scalds Among Children, J. Roy. Inst. Pub. Health & Hyg. **15**:32, 1952.

12. Kiskadden, W. S., and McDowell, A.: Immediate Treatment of the Burned Child, West. J. Surg. **58**:205, 1950.

13. Koch, S. L.: Use of Compression as a Surgical Principle in Treatment of Injuries, Quart. Bull. Northwestern Univ. M. School **17**:257, 1943.

14. Kyle, M. J., and Wallace, A. B.: Fluid Replacement in Burnt Children, Brit. J. Plast. Surg. **3**:194, 1950.

15. Moritz, A. R., in Symposium on Burns, National Academy of Sciences, National Research Council, Washington, D. C., 1951.

16. Prendergast, J. J.; Fenichel, R. L., and Daly, B. M.: Albumin and Globulin Changes in Burns as Demonstrated by Electrophoresis, A. M. A. Arch. Surg. **64**:733, 1952.

Nonpenetrating Abdominal Trauma

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Penetrating wounds of the abdomen imply underlying visceral damage, and surgical intervention is clearly indicated. Nonpenetrating abdominal injuries are equally lethal, but the seriousness of the injury may be underestimated. In the absence of visible signs of injury to the abdominal wall, the blunt force may be considered insufficient to produce visceral injury. Associated injuries may distract attention from the less obvious abdominal symptoms. A significant number of patients may have no pain or shock on admission to the hospital.[1] If an associated injury should require an emergency procedure, general anesthesia may mask the developing abdominal signs for many hours.[2]

The alarming increase in mortality resulting from delayed operation is pointed out in many reports.*

These observations prompted us to study the nonpenetrating abdominal injuries at St. Luke's Hospital for the years 1940 to 1954. The records of 47 patients were studied. Table 1 presents the distribution of injuries by organs involved. Injuries to the kidney and bladder were not included. Pancreatic injury was not found in this series, with the exception of a single minor contusion accompanying other, more major, injuries. Of the injuries of proved diagnosis, 36% involved the spleen, 36% the gastrointestinal tract, and 12% the liver, and 16% were multiple injuries, including three major vascular catastrophies. We have studied five "unproved cases" on the basis of strong clinical and laboratory evidence but without confirmation by operation or autopsy.

TABLE 1.—*Closed Abdominal Injuries (Forty-Seven Patients)*

Single Organ		35 (84%)
Spleen	15 (36%)	
Liver	5 (12%)	
Gastrointestinal	15 (36%)	
Jejunum and ileum	10	
Colon	3	
Duodenum	1	
Stomach	1	
Multiple		7 (16%)
Ileum, colon, superior hemorrhoidal, artery and vein	1	
Ileum, colon, superior mesenteric artery and vein	1	
Ileum, cecum, external iliac artery thrombosis	1	
Liver, small-bowel mesentery	2	
Liver, superior mesenteric artery and vein		
Small-bowel mesentery	1	
Pancreatic contusion	..	
Liver, inferior vena cava	1	
Unproved		5
Probable liver	1	
Probable multiple injuries with hemorrhage	1	
Probable intraperitoneal hemorrhage	3	

AGE AND SEX

The youngest victim of this series was aged 4 years, the oldest 63 years. Five patients were less than 20 years of age. There were 2 female and 45 male patients, probably because of the relatively low motor-accident toll and a proportionately large number of industrial accidents in this group. The report from the Birmingham[2] Accident Hospital indicates 15% incidence in females.

MECHANISM OF INJURY

The causes of abdominal trauma are not too varied. Sixty years ago, Curtis † re-

Read at the 63d Annual Meeting of the Western Surgical Association, Seattle, Dec. 1, 1955.

From the Department of Surgery, Northwestern University and St. Luke's Hospital.

* References 2, 3, 4, 5, and 6.

† Curtis, cited by Counsellor and McCormack.[3]

TABLE 2.—*Mechanism of Injury (Thirty-Nine Patients)*

Motor vehicle	13
Collision	1
Steering wheel	3
Squeezed	3
Struck by	6
Falls	10
Trains	5
Thrown from	1
Struck by	3
Squeezed by	1
Struck by blunt object	4
Squeezed by "Friend"	1
Squeezed between objects	3
Struck or crushed by falling object	3

ported that the most frequent cause was a kick from a horse. Today its successor, the automobile, is far and away the commonest cause. Many of these cases are due to steering-wheel impact. The second commonest cause is a fall. Patients in these two groups are seldom neglected. They are taken by ambulance to hospitals and receive the careful attention of the entire house staff. According to Welch and Giddings, 40% of nonpenetrating injuries fall into the category labeled "miscellaneous." Here lies the real danger. The trauma may be considered inconsequential and the true injury missed. Usually a small blunt instrument is the offender. A wheelbarrow handle, switch couple, or indoor baseball have all been frequent agents. Careful questioning of the patient, with an appreciation of the topographical anatomy of the abdomen, is essential.

Most authors emphasize the importance of unexpected injury. The recti muscles and fascia, when braced, have rebound and flexibility. When they are flaccid, they allow easy compression of the viscera against the unyielding bony column.

Obviously, the impact of a small blunt object can be expected to produce trauma which will be more localized than the rapid deacceleration occurring in vehicular collisions and falls from a height. The latter mechanisms will be responsible for many more "multiple organ" and associated injuries.

ASSOCIATED INJURIES

As stated above, the associated injuries are a result of a greater destructive force. The diagnostic problems may be multiplied. A patient whose conscious state is clouded by a head injury may not perceive abdominal pain. Fractured lower ribs on either side may give sufficient abdominal pain and spasm to mask the findings of an associated splenic or liver injury. Patients with a fractured lumbar vertebral transverse process may have received kidney trauma. The association of pelvic fractures with ruptured bladder is well known.[7] Greater judgment is required and a considerable outlay of professional skill required for successful treatment. Table 3 discloses the types of associated injuries encountered in 15 patients of this series. The 31 injuries listed attest to the severity of traumatic forces applied to these patients. Failure of treatment in this group is reflected by a mortality rate of 40%, as contrasted to the over-all rate of 23.4%. It is of interest that only one patient with intestinal perforation received extra abdominal injuries. This can be related to the local impact mechanism in nearly 60% of our isolated gastrointestinal injuries.

SYMPTOMS

All abdominal trauma is potentially serious, regardless of immediate symptoms. The

TABLE 3.—*Associated Injuries*

Total No.	Location of Abdominal Injury	No. of Patients	No. of Injuries	Cranio-cerebral	Spine, Sp. Proc.	Ribs	Pelvis	Extremities	Abdominal Wall	Diaphragm	Kidney
15	Spleen	8	17	1	1	6	2	4	1	1	1
5	Liver	2	2	..	..	2	..	..	..	..	..
15	Gastrointestinal	1	2	..	1	..	..	..	1	..	..
7	Multiple	3	6	2	..	1	..	2	1	..	..
5	Unproved	1	4	1	..	..	1	2	..	..	..
47		15	31	4	2	9	3	8	3	1	1

literature is filled with reports of delayed treatment of late recognized injury. Usually these are the fatal cases. The rapidity and severity of the onset of symptoms depend upon the location and severity of injury.

The immediate symptoms produced are those due to peritoneal irritation of a chemical nature, by gastric juices, bile, or intestinal contents, or to free blood due to rupture of liver, spleen, and omental or mesenteric vessels. Later or delayed symptoms are those of an infectious process.

Shock.—This is either present or impending, commensurate with the severity of injury.

Pain.—If a history can be accurately obtained, it is usually that of immediate abdominal pain following the injury; subsequently it may moderate, when the patient is put at rest and sedation administered. As soiling of the peritoneum advances, pain will again increase. The location of the original pain is indicative of the site of injury, an important point for the surgeon to record. After soiling of the peritoneum has become diffuse, pain as a localizing symptom is unreliable.

Pain referred to the shoulder or neck is indicative of diaphragmatic irritation due to either free air or fluids. Pain referred to the rectum and perineum with associated tenesmus is frequently a sign of high rectal or sigmoid injury. Pain may be referred to the right testis as a result of irritation of the genitofemoral nerve by spillage from retroperitoneal rupture of the duodenum. A very significant character of peritoneal pain due to spilling of either hollow visceral contents or blood is the exaggeration produced by body movement.

The pulse rate is increased with progressive soiling of the peritoneum. It is also increased in hemorrhage and shock and is therefore significant of serious intra-abdominal injury. Temperature may be misleading owing to the degree of shock present.

OBJECTIVE SIGNS

Rigidity and Tenderness.—The early picture of intra-abdominal injury is a silent, scaphoid, sensitive abdomen, depending upon the degree of trauma. The later signs of peritoneal contamination are those of distention, rigidity, and silent ileus.

Gastric Siphonage.—All cases of abdominal trauma should have gastric intubation. The presence of free blood indicates upper abdominal injury.

Rectal Examination. — The presence of fresh or darkened blood is indicative of intestinal injury.

X-Ray.—The presence of free air under the diaphragm is pathognomonic of hollow-viscus perforation. This is particularly true in perforation of the stomach or colon. Its absence does not exclude gastrointestinal perforation. Jejunal and ileal perforation seldom present intraperitoneal air. The small bowel seldom contains sufficient gas to allow x-ray recognition.

An exception must be made in retroperitoneal duodenal rupture, where retroperitoneal air may be seen to outline the psoas muscle shadow.

LABORATORY PROCEDURES

Urine.—The presence of blood in the urine should be sought as evidence of injury to the urinary bladder or kidney. The catheter, if used, can be left in place in order to follow the urinary output hourly as an aid to resuscitation. An output of less than 25 cc. hourly will probably indicate uncompensated shock or dehydration.

Blood.—The erythrocyte count is not dependable as an index of blood loss. Hemorrhagic shock was frequently present or impending in our spleen group with counts on admission ranging from 3,400,000 to 4,800,000.

Hemoglobin and hematocrit determinations, as a single determination, are equally misleading, and many hours with adequate hydration must elapse before the true level of cell dilution is reflected.

Of most value is the leucocyte count, which becomes rapidly elevated in most instances of intraperitoneal hemorrhage and as a result of chemical peritonitis from upper gastrointestinal spillage (average 20,000 on admis-

sion in ruptured spleens). Equivocal elevations or normal early counts must be discounted completely, if signs and symptoms of intra-abdominal injury are sufficient to justify exploration. Traumatic thrombosis of the vessels of the mesentery leading to subsequent gangrene and stripping of mesentery of limited small-bowel segments were instances in our group in which the leucocyte levels were misleading. Serial determinations will probably show definite trends, but usually clinical deterioration has preceded the laboratory evidence in the questionable case.

PATHOLOGY

The findings at operation and autopsy with respect to the liver ranged from superficial to deep lacerations involving chiefly the right lobe. In one case the gall bladder was torn (without rupture) from the liver bed. The dome of the liver and coronary ligament was involved in two patients.

Splenic injuries occurred as capsular tears to complete division of the lower fourth of the spleen (Fig. 1). There was no instance of complete avulsion of the spleen from its pedicle, corresponding to the fourth type of Blocker.[8] Nor was an instance of subcapsular hematoma, Blocker's Type 3, encountered. Two patients had so-called "delayed splenic hemorrhage" (McIndoe[9]), coming to operation 21 and 31 days after injury (Fig. 2).

The gastrointestinal lesions were observed as contusions, minimal perforations, extensive lacerations, and complete transections.

Fig. 1.—This illustration was drawn from the spleen removed from a 15-year-old boy who had been thrown from the platform of a street car, striking his left chest and abdomen. The diaphragmatic surface on the right presents a 5-cm. laceration extending through the inferior pole of the spleen. He was admitted one hour after injury in shock, operated in two and one-half hours, and recovered uneventfully.

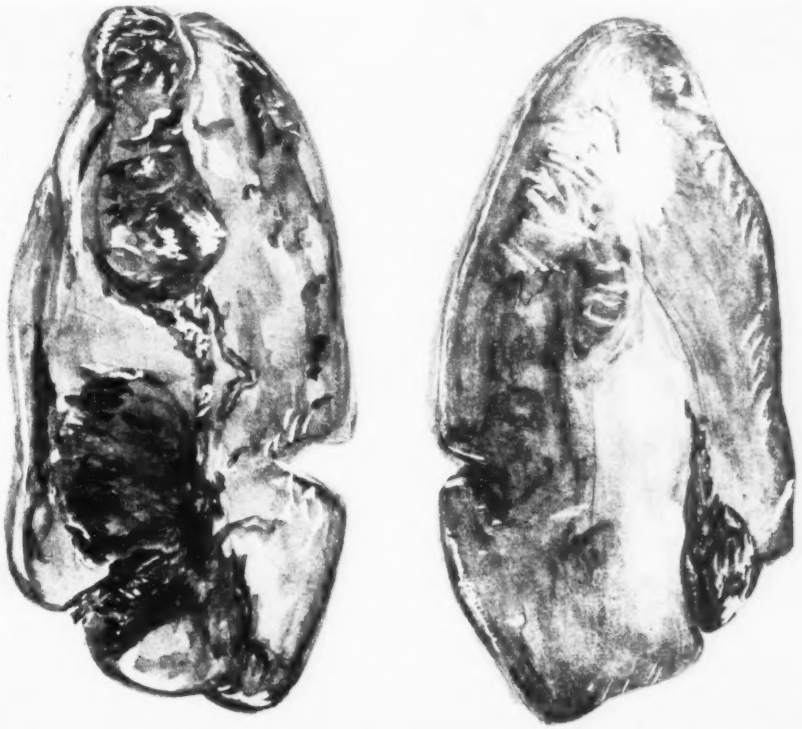

Fig. 2.—A 44-year-old man was admitted 30 minutes after having been struck by a car. He incurred fractures of the left clavicle, fifth, seventh, and ninth ribs, with only mild left upper-quadrant pain. On admission his blood pressure was 108/66; red blood cell count, 4,120,000; hemoglobin, 12.2 gm., and white blood cell count, 22,500. He was observed for 21 days. He developed hemothorax, a drop of the red blood cell count to 3,490,000, hemoglobin 10.2 gm., white blood cell count, 17,650, and a tender left upper-quadrant mass. Operation after this time disclosed a rupture of the anterior free margin of the spleen covered by a semiorganized cast of clotted blood with intervening fresh blood. The hilar surface appears on the left, diaphragmatic surface in the middle, and the blood cast of the spleen on the right.

In some instances the mesentery had been avulsed from the bowel without perforation. One of the cases operated upon late was found to have a traumatic thrombosis of the vessels of the descending colon with gangrene but without perforation. Deep tears into the mesentery were represented by divisions of the superior mesenteric vessels and tears of the superior hemorrhoidal artery (Fig. 3) and vein high in the mesentery.

PREOPERATIVE TREATMENT

There must be a prompt evaluation of the stage of the shock and an equally prompt treatment. All cases should have gastric intubation and gastric drainage prior to surgery. An indwelling bladder catheter is indicated. If the shock picture persists, surgical treatment to control the hemorrhage and continuing blood replacement may be the only successful method of reversing the shock syndrome.

OPERATIVE MANAGEMENT

The surgical incision should be sufficiently adequate and placed to allow easy access to all abdominal quadrants. A midparamedian incision, which can be extended superiorly or inferiorly, is the one of choice. Extension into the thorax could be useful. The frequency of multiple injury demands a complete abdominal exploration, with evaluation of the entire duodenum, pancreas, and diaphragm.

The simplest repair of intestinal perforation, which avoids constriction, is best for the hollow viscera. Resection should be done for extensive lacerations, transections, and

Fig. 3.—A pile of bricks fell, striking this patient from behind. He was folded over the tail gate of a truck. He incurred a compound frontal skull fracture, fracture of the left tibia, subcutaneous rupture of both rectus abdominis muscles, transection of the sigmoid colon, stripping of ileal mesentery, and division of the superior hemorrhoidal artery and vein near their origin. The drawing illustrates the surgical reflection of the left colon to gain proximal control of the hemorrhage. The aorta and superior hemorrhoidal origin from the inferior mesenteric artery are etched in behind the mesentery in the drawing. After arrest of hemorrhage, the small bowel was resected and anastomosed, and the colon ends exteriorized for later closure.

This patient recovered after a wound infection, but still has a diffuse lower abdominal hernia.

Fig. 4.—This drawing represents the effect of a car-coupling blow to the right lower quadrant of this railroad employe in 1945. Lacerations of the ileum and ileocecal mesentery necessitated resection and ileotransversostomy. There was also traumatic thrombosis of the external iliac artery and vein. In spite of procaine hydrochloride (Novocain) infiltration intra-abdominally to the sympathetic chain, and on subsequent days percutaneously, a midthigh amputation was necessary two weeks later. (The cut ends of the vessels in the drawing are schematic and do not represent a division of these vessels.)

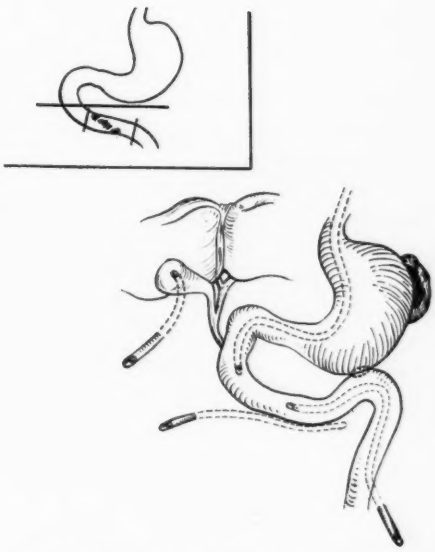

Fig. 5.—The drawing in the upper left represents retroperitoneal perforations of the duodenum, resulting from a contusion to the abdomen by "kick-back" of a file from a grinder. At operation 10 hours after injury, there was extensive air and fluid infiltration of the right retroperitoneal space extending from the liver to the pelvic brim. There was intense reaction and bile staining of all the periduodenal tissues, and the extent of duodenal injury seemed to preclude simple repair. The lower diagram represents the successful decompression of the duodenum and diversion of stomach contents by gastroenterostomy. The gall bladder was drained after evacuation of two stones to further divert bile from the duodenal injury. Positive suction was applied to the nasogastric, retroperitoneal, and jejunostomy tubes. It was thought that the gastroenterostomy would provide a satisfactory bypass in the event of a subsequent stricture. The patient recovered and has had no further trouble.

mesenteric vascular damage. In small-bowel cases primary anastomosis is mandatory, but the colon may be exteriorized for obstructive type resection and later anastomosis. Tears of the duodenum should be sutured, if possible. If suture is not feasible, the method employed in our case (Fig. 5) may

be chosen. A Whipple resection has been reported (Strode).[10] Siler's [11] cases illustrate the problem of treatment of duodenal rupture after varying time intervals. Rents in the mesentery must be closed to avert internal strangulations.

The spleen should be removed. After rapid determination of the damage, it is delivered from its bed as the most effective method for temporary control of the hemorrhage, whereupon deliberate isolation of the pedicle can proceed.

Liver injuries are best treated by mattress suturing with wet plain absorbable surgical (gut) sutures or absorbable gelatin sponge (Gelfoam) sutured into the defect. Resection of part of a lobe may be necessary. Drainage is nearly always desirable to conduct any leaking bile from the abdominal cavity.

Adequate toiletry and, in general, primary closure is the procedure of choice. The simplest and fastest abdominal wall closure in the face of shock is desirable.

POSTOPERATIVE TREATMENT

The essentials at this stage are those of general postoperative care, with particular emphasis on restoring a normal blood and red-cell volume. Repeated hematocrit readings for the ensuing 72 hours [12] are the best guide for blood requirements. Patients should not be allowed to struggle along in a state of compensated shock. Nothing favors prompt healing and restoration of a normal physiology as much as blood, adequate fluids, and electrolytes.

An aggressive program toward avoidance of pulmonary complications should be carried out, i. e., (1) cough regimen, (2) intercostal procaine hydrochloride (Novocain) block, (3) emulsant therapy, (4) bronchoscopic aspiration, and (5) prophylactic tracheotomy. Nasogastric suction and appropriate antibiotics are indicated.

COMPLICATIONS

Many of these severely injured patients have a stormy postoperative course. Peritonitis and wound infections are not uncommon and can be minimized only by early adequate surgery. No eviscerations were counted among our patients. Several had postoperative hernia requiring later repair. Secondary mechanical intestinal obstruction occurred twice. These patients were relieved by surgical therapy. Frequent incidence of atelectasis, nonfatal and fatal, prompted us to stress the preventive and therapeutic pulmonary regimen under postoperative care.

MORTALITY

The deaths in this group are explained in Tables 4 and 5. Although the majority could be considered justified by the severity of injury, we cannot relent in the immediate shock therapy or aggressive operative attack until an insurmountable situation is proved. Promptness of transportation to the hospital is shown in the patients who were alive to undergo exploration with such injuries as divided superior mesenteric vessels and torn vena cava.

TABLE 4.—*Deaths (Cases Without Operation)*

Location of Injuries	No.	Time	Cause of Death
Unoperated			
Spleen			
Multiple ribs and pelvis	1	2 hr.	Shock and hemorrhage
Comatose	1	15 min.	Shock and hemorrhage
Liver			
Multiple rib fracture	1	1 day	Shock and pulmonary
Multiple rib fracture plus cerebral concussion	1	2 days	Not recorded
Multiple			
Liver and inf. vena cava laceration	1	35 min.	Hemorrhagic shock
Liver laceration, mesentery small bowel, and multiple rib fracture	1	3 days	Hemorrhage-atelectasis
Unproved			
Craniocerebral injuries, fractured pelvis, and extremity fractures	..	Within 1st 4 hr.	Multiple injuries, shock, and hemorrhage

TABLE 5.—*Deaths (Cases with Operation)*

Location of Injury	No.	Time Operated	Time Death	Cause of Death
Spleen				
Fractured 6 to 10 left ribs, left tibia, and fibula, possible skull fracture	1	9 hr.	48 hr.	Atelectasis
Gastrointestinal				
Jejunum and ileum	1	3 days	.....	Peritonitis
Jejunum and ileum	1	3 days	6 days	Peritonitis
Colon; division superior mesenteric artery and vein	1	3½ hr.	1 day	Massive gangrene small and large bowel
Multiple				
Lacerated liver and superior mesenteric artery and vein division; fractured fibula	..	6 hr.	11 hr.	Massive gangrene small and large bowel

TABLE 6.—*Time Interval to Operation and Mortality in Bowel Injuries*

Time, Hr.	No. Patients	Results
Less than 7	9	No deaths
54	1	Peritonitis, survived
72	2	Peritonitis, 2 died

The time interval to operation of the bowel injuries is compared with the mortality in Table 6. A similar analysis was presented by Poer [4] in 1941. Our results, when operation was early, are an improvement, but the late cases are remarkably the same.

Table 7 presents the percentage calculations of mortality in relation to treatment as well as the over-all mortality, namely, 23.4% for the entire series.

COMMENT

This study has again emphasized the severity of trauma which can be withstood for even a short time by the human organism. As first aid and ambulance service improve, a greater number of patients arrive for hospital care with injuries in the past considered to be mortal. This proportion may vary with the location and practice of the hospital. We are obligated to provide prompt and sufficient resuscitation while evaluation of the injuries is in progress and to proceed vigorously and intelligently with appropriate operative treatment.

The challenge is (1) recognition of the presence of visceral injuries and hemorrhage, (2) keeping the patient alive by resuscitative measures, and (3) repair of the injured structures.

In the presence of associated injuries (especially to the thorax), the only relatively safe opportunity for a general anesthetic will be early. Certainly massive hemorrhage can only worsen the condition of the patient, as will developing peritonitis, should a waiting policy be adopted.

In the absence of any completely reliable x-ray or laboratory diagnostic procedures, the final responsibility for bringing the patient to early exploration still rests with

TABLE 7.—*Nonpenetrating Abdominal Visceral Injuries at St. Luke's Hospital (1940-1954)*

Viscus	Patients, No.	Treated Surgically			Treatment Expectant			Total Mortality, %
		No.	Deaths	% Mortality	No.	Deaths	% Mortality	
Spleen	15	13	1	7.6	2	2	100	20
Liver	5	4	0	0	1	1	100	20
Jejunum ileum	10	10	2	20	0	0	0	20
Colon	3	3	1	33.3	0	0	0	33.3
Duodenum	1	1	0	0	0	0	0	0
Stomach	1	1	0	0	0	0	0	0
Multiple	7	5	1	20	2	2	100	42.8
Unproved	5	0	0	0	5	1	20	20
Totals	47	37	5	13.5	10	6	60	23.4

the clinical judgment of the surgeon. A small percentage of negative explorations would seem preferable to mortalities from missed diagnosis. Equally as important as the preoperative resuscitation is the anesthesia. It would seem appropriate to explore the usefulness of hypothermia as an adjunct to the anesthesia for the more seriously injured of this group. To avoid delay, the cooling can be carried out along with resuscitation. The anoxic damage to the vital organs should be lessened, and the ability of the patient to withstand a long reparative procedure should be improved.

Repair, or replacement by graft, of major vessels, such as the vena cava, superior mesenteric artery, and the iliac artery, must be considered. Although these procedures have been accomplished more frequently in penetrating injuries, Ulvestad [13] in 1954 reported the successful repair of the superior mesenteric artery injured by blunt trauma.

Mortalities cited in the literature must be carefully evaluated with respect to types of injury, size of the series, number of cases proved by operation or autopsy, and incidence and kind of associated injuries. The time to admittance will produce a certain selectivity, i. e., where prolonged, the most severely injured will not survive transportation to the hospital. Taking such facts into consideration, we present a few representative figures.

Poer and Woliver [4] (1942) adding to the compiled figures of Counsellor and McCormack (1935), counted 1476 cases of gastrointestinal and mesenteric injury in 1014 of which operation had been done. Operative mortality was 50.4%; total mortality, 61.3%.

From the Massachusetts General Hospital, Welch and Giddings (1950) reported 200 cases of abdominal trauma. Of these 45.5% were kidney injuries, and 86% were nonpenetrating injuries. The total mortality in these 172 cases was 13.6%. Estes, Bowman, and Meilicke [5] (1952) analyzed 67 cases (18 kidney) and found a 19.4% total mortality. Of 273 cases studied by Starkloff [6] (1953) at St. Louis City Hospital only 28% were nonpenetrating. Total mortality was 15%, operative mortality 13.2%. (Forty-seven patients had negative explorations.) Clarke,[2] of Birmingham, published a study of 120 cases in which 107 were proved by operation and 13 by autopsy. The operative mortality was 22.4%.

All of the recent studies indicate improvement over the earlier experience and denote the degree of interest in these unfortunate injuries. One cannot but hope that accident prevention will make greater strides for the benefit of those who may have been destined for the dwindling but more difficult "mortal injury" group of the future.

SUMMARY

Records of 47 patients presenting nonpenetrating trauma have been studied and discussed relative to mechanism of injury and associated injuries. Symptomatology and objective findings, pathology, and treatment are considered. Illustrative cases are presented. The deaths have been analyzed. Mortality related to time of operation and over-all mortality are presented. The mortality figures from recent reports in the literature are mentioned, and variations pertaining to mortality figures are pointed out.

CONCLUSIONS

Improvement in the mortality in patients with abdominal trauma will depend on (1) earlier admittance to the hospital, (2) rapid restoration of blood loss, (3) early exploration on the basis of reasonable suspicion of isolated intra-abdominal injury, (4) careful evaluation of abdomen in presence of multiple injuries, (5) use of modern restorative vascular techniques, (6) vigorous treatment of air passages in associated chest injuries, and (7) appropriate antibiotic and electrolyte therapy.

DISCUSSION

DR. EDWIN M. MILLER, Chicago: I want to make only two points.

The first is that these nonpenetrating abdominal injuries are real emergencies, and they take priority. We speak of operations for appendicitis as being emergencies. This type of case is a real emergency, allowing no delay whatever if we are going to de-

crease the frightful mortality that attends some of these injuries.

The second thing I want to say is that one should never forget in any case to explore the retroperitoneal part of the duodenum, regardless of whether the patient is suffering from obvious serious injury elsewhere, because it is in these neglected cases of retroperitoneal perforations of the duodenum that we see almost 100% mortality.

This was emphasized many years ago, and one of those who took great pains to bring this out was Dr. Allen Knarel, of Chicago. Because of its anatomical position and relationship, sometimes the duodenum is almost a closed loop between the pylorus and the third part of the duodenum, where it is overlaid by the mesentery. Potentially it is a closed loop, and when distended with fluid and when force is applied suddenly in a patient who is in a relaxed state, let us say, not anticipating injury at all, when there is no defensive mechanism set up, one may get the full force of the blow exerting itself as an explosive type of injury to the duodenum, thus producing a leak in the retroperitoneal part that might not be manifest clinically or might not be manifest even on first glance as one opens the abdomen.

So, I would say that in every case, no matter how obvious the injuries are elsewhere in the abdomen, one should never forget to explore very carefully for the telltale signs of a retroperitoneal perforation of the duodenum.

Dr. Chester C. Guy, Chicago: I have enjoyed this interestnig and valuable paper by Drs. Pontius, Kilbourne, and Paul. I presume one of our greatest diagnostic problems in patients with real or suspected blunt injury to the abdomen is in those cases where we find multiple injuries, when the patient is perhaps unconscious from a head injury, has fractures, possibly serious ones to the extremities, and the question is raised as to whether there is or is not intra-abdominal trauma.

These, it seems to me, constitute as difficult a problem in the handling of injured patients as any we are called upon to see.

I should like to emphasize particularly two lesions, one of which has been covered by Dr. Miller. The first is the delayed rupture of the spleen. In a survey some years ago of a series of cases of delayed or secondary rupture of the spleen, we found that this may occur as long as two to four months after the original injury. The majority, however, occur within the first two weeks.

I should like to make a plea that patients who have left upper quadrant or left lower chest injuries, especially if there are fractures of the 10th and/or 11th ribs, be carefully kept under observation for a period of at least two weeks.

Many of the deaths from secondary splenic hemorrhage have been in patients in whom the attending surgeon has forgotten the left upper abdominal injury because his attention has been directed perhaps to other injuries. Then the patient suddenly has an intra-abdominal hemorrhage, and the etiology of it may not be suspected. A great majority of those patients will die if they are not promptly and properly operated upon.

The second point (which Dr. Miller so nicely covered) has to do with the exploration of the retroperitoneal portions of the duodenum after upper abdominal injury. I have seen four cases of retroperitoneal rupture of the duodenum when there were no other injuries (at least of any consequence) to the abdomen. Two I have operated on, one of which recovered, and I noted with interest the ingenious method that the authors used to handle the problem in the case they had.

I have seen two retroperitoneal ruptures of the duodenum at autopsy.

[Slide] This is a photograph of an autopsy specimen from a young Negro man who fell a distance of 8 ft. and struck the right upper quadrant of his abdomen. He was immediately taken to a hospital, where his symptoms did not seem to be severe. He appeared to be not too seriously injured under rather casual observation during the next 12 to 14 hours. About 20 hours after his injury, exploration was done by a surgical resident because he then complained of lower abdominal pain and also pain in his right loin. He went into shock on the table. The resident confined his exploration to the lower half of the abdomen and did not even explore the upper half. The patient died about 30 hours after the injury, and the autopsy revealed these two longitudinal tears in the second portion of the duodenum. There was no peritoneal tear and no peritonitis. Injury was due to a retroperitoneal phlegmonous infection.

As has been pointed out, these retroperitoneal tears will always result fatally if they are not surgically treated, and to be treated properly by surgery the surgeon must actually look for them, for otherwise they can easily be overlooked.

Dr. Robert A. Wise, Portland, Ore.: The inclusion of four papers on the surgery of trauma in this interesting program this morning attests to its vital importance. Any disease process which accounts for 100,000 deaths a year in this country and 10,000,000 injuries certainly calls for our very serious thought and study.

I was tremendously interested in this paper on nonpenetrating abdominal trauma. I should like to second the remarks made by Dr. Miller and by Dr. Guy concerning the rupture of the retroperitoneal portion of the duodenum. We had such a situation several years ago. A young man fell from a horse. The horse then fell upon him, driving the pummel of the saddle against his upper abdomen. On exploration several hours later, there was a diffuse peri-

tonitis but there was no apparent perforation. Upon explorating the upper abdomen, the telltale bile discoloration of the posterior peritoneum with edema was present.

[Slide] There was almost complete rupture of the third portion of the duodenum, only a small section of the posterior wall remaining intact. It was not difficult to perform an end-to-end anastomosis of this transection of the duodenum.

I should like to bring out one other point about perforation of an abdominal viscus from nonpenetrating trauma. One particular characteristic of the condition is the frequent delay in the development of signs of an acute abdominal condition. The delay in signs may be due to a temporary closure of the perforation either by omentum or by prolapsed mucosa or by an actual delay in the development of the perforation which at times is due to bowel-wall ischemia secondary to injury of the mesenteric vessels.

[Slide] This point is illustrated by a patient with a perforation of the jejunum 8 cm. from the ligament of Treitz due to nonpenetrating abdominal trauma incurred in an automobile accident. He walked away from the accident and was taken in the car of a passing motorist to a local hospital. He complained of no abdominal pain, and this patient was discharged from the hospital the following morning. It was not until 24 hours later, upon entrance to the Veterans Hospital, that unmistakable signs, including free peritoneal air, were present. It was an easy matter to suture this perforation.

Dr. William R. Lovelace II, Albuquerque, N. Mex.: I should like to point out a couple of factors that can cause this type of injury which Drs. Pontius, Kilbourne, and Paul did not bring out.

I enjoyed their presentation very much. As many of you know, airplanes are traveling at extremely high speeds, and some of the men now are having to bail out by use of an ejection seat while traveling 700 to 800 miles an hour. When that happens, there is a wind or blast pressure of approximately 13 lb. per square inch applied to the body, resulting in tremendous compression with possible intra-abdominal and thoracic injuries. Flailing of the extremities and neck can also occur.

Another factor is that if these men are ejected and have to open their parachutes quickly because of closeness to ground they can have a force applied to them of up to 6,000 to 8,000 lb. from the opening shock and still live. We have had several cases in the Air Force when they have sustained injuries to the liver, gall bladder, and intestines.

Another source of trauma is that of blast injury that occurs when an atomic bomb goes off. We have been carrying out experimental work on this. There is a wind speed of several hundred miles an hour in a certain area, which can be damaging in itself. The blast also results in secondary missiles that can strike a person and can result in very severe nonpenetrating as well as penetrating abdominal injuries.

Dr. Carleton Mathewson Jr., San Francisco: I should like to emphasize three points.

First, very mild trauma to the abdomen may and often does cause very extensive and serious injury. This fact should not be overlooked.

Second, when you do explore an abdomen after blunt injury, you should proceed just as you do after penetrating wounds of the abdomen; that is, make an adequate incision so that you can carry out a carefully planned exploration.

We have a patient in the hospital at the moment who fell on her side from a height of three stories. No serious injury other than an abdominal one was found. She had four blowouts in the small intestine in unattached areas of the jejunum. One of these was almost overlooked because it was so very small and located on the mesentery side of the bowel. One should look very carefully and explore the entire abdomen whenever one suspects an injury.

The third point I should like to make and emphasize is that the first doctor who sees the patient should establish a base line by making a very careful record of the abdominal findings at the initial examination, because from there on out changing signs are the ones that help us establish a diagnosis.

Dr. Burton C. Kilbourne, Chicago: I wish to thank Dr. Pontius and the members for the invitation to attend this meeting. I appreciate the discussion, and the points brought out by Drs. Miller, Guy, Wise, and Mathewson.

Dr. Wise's patient, presenting a perforation of the jejunum without immediate distressing symptoms, impresses me as being quite unusual. Ordinarily, the chemical irritation of jejunal contents is much the same as that of the stomach contents, and the instances of perforation that we have observed presented rather marked pain with board-like rigidity. Soilage by contents of the large bowel is less irritating, and the onset of pain is frequently delayed. In the instance when the mesentery was contused and secondary thrombosis occurred without perforation, the patient was observed for two or three days before signs of infection and gangrene appeared.

REFERENCES

1. Welch, C. E., and Giddings, W. P.: Abdominal Trauma: A Clinical Study of 200 Consecutive Cases from the Massachusetts General Hospital, Am. J. Surg. **79**:252 (Feb.) 1950.

2. Clarke, R.: Closed Abdominal Injuries, Lancet. **267**:6844 (Oct. 30) 1954.

3. Counsellor, V. S., and McCormack, C. J.: Subcutaneous Perforation of Jejunum, Ann. Surg. **102**:365 (Sept.) 1935.

4. Poer, D. H., and Woliver, E.: Intestinal and Mesenteric Injury Due to Nonpenetrating Abdominal Trauma, J. A. M. A. **118**:11 (Jan. 3) 1942.

5. Estes, W. L., Jr.; Bowman, T. L., and Meilicke, F. F.: Non-Penetrating Abdominal Trauma: With Special Reference to Lesions of Duodenum and Pancreas, Am. J. Surg. **83**:434 (March) 1952.

6. Starkloff, G. B.; Bailey, D., and Rutledge, R.: Abdominal Trauma, Missouri Med. **50**:918 (Dec.) 1953.

7. Baker, W. J., and Graf, E. C.: Management of Urinary Tract Injuries in Fracture of the Bony Pelvis, S. Clin. North America **35**:295 (Feb.) 1955.

8. Blocker, T. J., Jr.: Traumatic Rupture of Spleen, Texas J. Med. **35**:478-483 (Nov.) 1939.

9. McIndoe, A. H.: Delayed Haemorrhage Following Traumatic Rupture of the Spleen, Brit. J. Surg., Nov. 1939, Vol. XX, page 249. **20**:249 (Nov.) 1939.

10. Strode, J. E., and Gilbert, F. I., Jr.: Retroperitoneal Rupture of Duodenum Following Nonpenetrating Injuries to Abdomen, A. M. A. Arch. Surg. **70**:343 (March) 1955.

11. Siler, V. E.: Management of Rupture of Duodenum Due to Violence, Am. J. Surg. **78**:715 (Nov.) 1949.

12. Stanton, J. R.; Lyon, R. P.; Freis, E. D., and Smithwick, R. H.: Blood and "Available Fluid" (Thiocyanate) Volume Studies in Surgical Patients, Surg. Gynec. & Obst. **89**:181 (Aug.) 1949.

13. Ulvestad, L. E.: Repair of Laceration of Superior Mesenteric Artery Acquired by Non-Penetrating Injury to Abdomen, Ann. Surg. **140**:752 (Nov.) 1954.

Spinal-Cord Injuries

A Fifteen-Year Survey

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The subject to which we have addressed ourselves is a difficult one at best because the accumulation of reliable data in sufficient volume is a major undertaking; the occurrence of comparable cases from which to draw conclusions is infrequent, since the morbid physiology of the lesions is dynamic; follow-up studies are difficult, and in those cases in which the patients die autopsies may be refused, and in those cases in which the patients live the neurological analysis may be confused by related conditions, such as infection. Nonetheless, the very difficulties inherent in the situation demand that an effort be made to survey again critically the present state of therapy of spinal-cord injuries. The present study is an effort to clarify our own thinking in relation to the experience of one of us (J. P. E.) over the 17-year period (1937-1954) when he was in charge of the Neurosurgical Service at the Cincinnati General Hospital. We hope by this report to renew interest in and stimulate discussion of a problem of considerable importance. Our conclusions in regard to certain diagnostic maneuvers and therapeutic procedures may not pass unchallenged.

We have reviewed the records of the Cincinnati General Hospital from 1938. The total number of cases whose charts were reviewed is 87. From the data gathered we have sought to obtain the answers to several questions which will be proposed later, below.

Read at the 63d Annual Meeting of the Western Surgical Association, Seattle, Dec. 1, 1955.

From the Division of Neurological Surgery, University of Chicago Clinics.

Of the 87 cases 79 were admitted to the General Hospital immediately after injury. The remaining eight cases were admitted late either for other reasons or for the specific purpose of treating the sequelae of an old spinal-cord injury originally treated elsewhere. In the total group are included seven cases of cauda equina injury.

For the purposes of the present analysis these last cases will be excluded from the discussion, though not from the statistical data. Of the 43 patients with cervical-cord injuries, 51% died during hospitalization; of the 29 with thoracic (dorsal)-cord injuries, 31%, of those with caudal lesions, none. Despite the fact that these are civilian hospital cases, one-third represented gunshot (21) and stab (6) wounds.

A chronological division of the fatal cases was arbitrarily made into three groups. The first includes those dying in the first 24 hours following admission, and in general they include those cases suffering severe associated injuries, such as penetrating chest wounds, multiple extremity injuries, ruptured bowel, etc. Five of the thirty-one fatalities occurred in this early period.

The second group includes deaths occurring from the second through the sixth day, inclusive. This is the period when maximum cord swelling presumably occurs. As would be expected, cervical injuries predominate in this group, which includes 11 cervical cases and only 4 thoracic injuries; of these latter cases two were high thoracic gunshot wounds. The other two had severe associated injuries.

As might be anticipated, life expectancy following the first week increases sharply, and of patients with cervical injuries surviving this period 75% may be expected to leave the hospital alive, and of those with thoracic-cord injuries, 84%. Looked at from the

standpoint of rehabilitation, it may be said that, though only 57% of all with spinal injuries who enter a hospital will survive, 79% of those surviving the first week of hospitalization will be candidates for rehabilitation. The high fatality rate in the first week is due, of course, to the relatively greater frequency of cervical injuries (43 to 29 in this series) as well as to the greater threat accompanying cervical injury.

Faced then with this grave situation, we need to ask ourselves certain questions concerning the value of our diagnostic maneuvers and the efficacy of our therapeutic procedures.

One of the first questions which we asked of ourselves is the following: "What is the significance of cerebrospinal fluid block on which the decision for or against operation is so frequently based?"

In reviewing our material we found that in those patients who subsequently died the test was for all practical purposes an unimportant criterion, because (1) it is occasionally misleading, since in two of the instances in which block was demonstrated operation revealed no swelling; (2) in many patients in whom block is demonstrated the patient's condition is too serious to permit operation, and (3) in certain instances lumbar puncture is impractical in the face of associated injuries, such as rib-cage fractures.

In the 21 surviving patients with cervical-cord injuries there were 3 with demonstrated block who were not operated on. In these three cases it is proper to ask the question whether operation might have improved the situation. For these specific cases a specific answer is difficult, but we have concluded, comparing the degree of recovery in those cases without block, that there is little reason for believing decompressive laminectomy would have helped the more seriously injured and blocked cases.

Of the four patients with thoracic-cord lesions who survived and on whom after the demonstration of manometric block an exploratory procedure was done, there seems to us no valid evidence that the results could have been worse had the operation and the antecedent procedure, lumbar puncture, not been done.

There were two cases of thoracic injury in which manometric block was demonstrated but laminectomy was not carried out. Again one must raise the question whether these cases might conceivably have been benefited by operation; but again there is grave doubt in comparison with the results obtained in nonblocked cord injuries not operated upon that this would have been of benefit.

From the therapeutic point of view the obvious crucial question is "Does laminectomy improve the prognosis in spinal-cord injury?" The proper answer would require two parallel series gathered alternately with and without laminectomy. This manifestly is a doubtful possibility on any clinical service. The best approximation would be careful analysis of a large series of cases—much larger than our own.*

Of our 21 surviving cervical cases, only 5 were subjected to laminectomy, and, since 1 had an ice-pick stab wound and another a gun-shot wound, we are left with only 3 cases. The first of these was a fracture dislocation at C-4 and C-5 in which there was no block and in which there was no improvement at the time of discharge, four and one-half months after the injury. A second case of fracture dislocation at C-5 and C-6 was operated upon late and fused but showed no improvement. Lumbar manometrics were not determined in this patient. The third case, a cord contusion resulting from C-2 and C-3 dislocation, showed a manometric block but had no benefit at the end of three and one-half months following operation.

Of our 20 dorsal cases 10 were subjected to laminectomy, and, since 5 of these were for gunshot wounds, we are left with only 5 cases for consideration. In one of these 5 cases cord injury was mild, manometric

*Fortunately such a series dealing with some 500 cases is presently being gathered by Dr. Jack French from the files of the Veterans Administration Hospital, Long Beach, Calif.

responses were not tested, and the exploration was negative. In another without manometric studies, a case of compression fracture at T-12 who had an excellent recovery, laminectomy was not done until late, at which time the patient underwent a fusion and exploration was purely incidental. Of the remaining three, the two who had block showed virtually no recovery and the final patient was operated upon without manometric tests and had virtually no recovery.

These cases are too few in number to allow one to draw any firm conclusions, but there is in these specific cases no clear indication that laminectomy has been of any significant value.

One is led then to the following question, "Why has laminectomy been suggested as an effective means of improving cord function following trauma?" The indications may be listed as follows: (1) to remove foreign bodies that are in the spinal canal, e. g., bullets; (2) to remove compressing bone fragments or to correct dislocations that are judged to have a compressing effect; (3) to remove protruding intervertebral discs; (4) to evacuate extradural hematoma; (5) to relieve spinal-cord swelling, as suggested by the experimental work of Allen,[1] and (6) to relieve vascular embarrassment.

COMMENT

The following critique of these reasons is based on our own clinical experience and the review of the cases included in the present series.

1. *For the Removal of Foreign Bodies Lying in the Spinal Canal.*—On the basis of experience in one case, in which a .22-caliber bullet came to lodge in the ligamentum flavum over the dorsal surface of the cord, we should conclude that such a lesion may cause significant cord compression unassociated with spinal block and that such a foreign body should be removed.

2. *For the Removal of Compressing Fragments of Bone.*—In the present series we have no evidence which bears upon this question, but indirect argument from the case cited immediately above would lead us to make the effort to restore the normal architectural relationships of the spine. It should be added, however, that of the three cases of cervical dislocation treated by laminectomy, one without block and two with block, there was obtained no significant benefit. In the dorsal region there were three dislocations, one with block and two not tested. Among these there was no significant benefit in two, and one was operated upon late and was fused and, therefore, what improvement was noted presumably antedated the operation. We must conclude then that in this very small series laminectomy was not of benefit.

3. *For the Removal of Protruding Intervertebral Discs.*—In our entire group, including postmortem cases, there has, to the best of our knowledge, been only one case in which protruding disc as a single entity in the total picture was of particular importance. This was an instance of cervical injury with the development months later of signs that suggested a median disc protrusion. In this case spastic quadraparesis developed three months after the dislocation and at the time seen (1948) exploration was not done.

4. *For the Removal of Extradural Hematomas.*—Only two cases of extradural hematoma were found in this series (including 15 operations and 13 postmortem examinations). Both of these were discovered at autopsy performed upon patients who had suffered extremely severe injuries. One of us (J. P. E.) has, in his own experience, never seen at exploratory laminectomy in traumatic cases an instance of extradural hematoma.

5. and 6. *For Relief of Spinal-Cord Swelling and Vascular Embarrassment.*—We need next to consider the assumption that swelling can develop sufficiently to interfere with blood supply and as a corollary that dorsomedian raphe incision as suggested by Allen may be of value. In accepting this argument, one must conclude that the pia has no, or at most inconsequential, capacity for stretch. So far as we know, there is no conclusive evidence on this point. While, on the one hand, we would argue that the force with which damaged spinal-cord tissue extrudes from an opening in the pia suggests that the

pia does not stretch, one must recognize that the pressure demand is great. One of us in conjunction with Scheinker has studied a case of cervical-cord injury in which the post-mortem studies seem to leave no doubt but that swollen cord substance had extruded through a dural decompression and therefore the pia arachnoid must have stretched. Undoubtedly distention of the jackets occurs in chronic expanding lesions, but we have little evidence of such distensibility in acute lesions. It seems to us that the question must for the time being be left open. What has been said for the pia holds likewise for the arachnoid.

That the dura ever constricts an acutely swollen cord is open to question, though one case in our own experience suggests that this may be the case.

From the practical point of view, however, were effective decompression to be obtained, pial incision is an essential, and any of us who has seen the cord extrude at operation when the pia is incised cannot but shudder at the thought of tract and gray-matter disruption that must occur as cord substance pours out like paste from a tube.

It is then evident that we are skeptical of the practical applicability in humans of Allen's experimental work on cord injury. Actually, in the experimental design of Allen an artificial situation was created, since the force delivered to the cord by falling weights was transmitted to an area of the cord already subjected to laminectomy. While it may be a purist's argument to say that he was not studying a parallel problem, the fact remains that the circumstances of the usual cord injury in humans were not duplicated.

We are not, as yet, prepared to make a practical application to our problem of Tarlov's [2] conclusions concerning cord compression. It would seem, however, that the demonstration of the effects of acute ischemia and of the brief periods of time in which irreparable damage is accomplished suggests that the scalpel provides assistance too little and too late.

In conclusion then, and rather to our surprise, our survey leads us to believe that spinal manometric studies are of virtually no practical value in making a judgment concerning operative interference. Further, and to our great distress, we have been able to adduce no positive evidence that decompressive laminectomy has been of value in those cases which we have studied. There are several instances above of cervical and thoracic lesions in which one may justifiably raise the question, "If laminectomy and decompression had been done, would the patient's chances of improvement not have been enhanced?" Comparison with presumably somewhat less injured cases, who did not demonstrate block but whose over-all problems otherwise seem comparable, suggests that operation would not have helped, for in the latter group recovery was also poor.

We dislike to end this discussion on a pessimistic note, but, quite apart from the tremendous improvements that have been accomplished in the handling of the problems of the paraplegic patient, we find little about which to be encouraged. Likewise, until a much clearer understanding of the physical chemistry of nervous-tissue swelling is achieved, we do not see any likelihood of substantial hope for better handling of the primary neural damage. This, then, is but a part of a very fundamental problem demanding much further basic research in the physiology and pathology of central-nervous-system lesions.

DISCUSSION

DR. DAVID L. REEVES, Santa Barbara, Calif.: I enjoyed this paper by Drs. Evans and Rosenauer very much. From my own personal experience during the last war I can go right down the line in agreeing with them.

We all know that there are cases of concussion of the cord in which the paraplegia may completely disappear in a matter of three or four days to a few weeks. We also know that there are patients with contusions of the cord of mild varieties who recover almost completely in a matter of weeks. I believe that in those cases that might have been subjected to laminectomy the laminectomy would have been given credit for the end-result.

Obviously there is often a great deal of pressure brought to bear on the surgeon to do an immediate laminectomy in these cases that are so distressing. Certainly at times it is difficult to withhold that

procedure, and I am sure many of us are forced to operate when we realize that the beneficial effect will not be forthcoming.

In many instances a laminectomy is done later because the patient feels that he would like to have the cord explored to be certain that everything possible has been done to assure him of the fact that nothing further in the way of recovery may ensue, and in many veterans' hospitals just after the war this was the procedure, and it was presented to the patient. If he so chose, then a laminectomy was done.

I certainly appreciate the importance of this paper, and I agree wholeheartedly with everything that Drs. Evans and Rosenauer have said.

DR. JOSEPH P. EVANS, Chicago: In those instances when one may occasionally be pressed to do a laminectomy, one really should do it with his eyes wide open to the probability that he is accomplishing nothing more than reassurance for the patient that everything possible has been done, as Dr. Reeves has pointed out.

I believe that this may be a point we should think about seriously. Recently there has been occasion to discuss this with a physician, also a neurosurgeon, who himself had suffered transection of the cord for other reasons. After he talked about the problems that a paraplegic has to face, he said he felt that in many instances, for the sake of the patient and the patient's family, operation might have to be undertaken, but again if one has to do it on that basis he should have his eyes wide open to what is going to result.

ADDENDUM

After the preparation of the galley proof of our article, the report of the large series of cases from the Veterans Administration Hospital at Long Beach alluded to in our footnote has been published: Comarr, A. E., and Kaufman, A. A.: A Survey of the Neurological Results of 858 Spinal Cord Injuries: A Comparison of Patients Treated With and Without Laminectomy, *J. Neurosurg.* **13:** 95-106, 1956. This article should be seen, for it expresses conclusions divergent from our own.

REFERENCES

1. Allen, A. R., in Frazier, C. H.: Surgery of the Spine and Spinal Cord, New York, D. Appleton & Co., 1918.

2. Tarlov, I. M.; Klinger, H., and Vitale, S.: Spinal Cord Compression Studies: Experimental Techniques to Produce Acute and Gradual Compression, A. M. A. Arch. Neurol. & Psychiat. **70:**813-819, 1953.

3. Tarlov, I. M., and Klinger, H.: Spinal Cord Compression Studies: Tissue Limits for Recovery After Acute Compression in Dogs, A. M. A. Arch. Neurol. & Psychiat. **71:**271-290, 1954.

4. Tarlov, I. M.: Spinal Cord Compression Studies: Tissue Limits for Recovery After Gradual Compression in Dogs, A. M. A. Arch. Neurol. & Psychiat. **71:**588-597, 1954.

Soft-Tissue Tumors of the Extremities Exclusive of Skin and Lymphatics

RALPH K. GHORMLEY, M.D., Rochester, Minn.

The subject of soft-tissue tumors of the extremities, exclusive of epithelial tumors, has not been accorded widespread recognition and discussion for several reasons. Except for the contributions of Stout,[1] Pack,[2] Cade,[3] and Jönsson,[4] relatively little has been offered in the literature in the way of information. Stout,[1] particularly, has written extensively regarding these tumors and has done much to develop a working classification of the group.

These tumors involve mainly the connective tissues and arise from cells derived from the mesenchyme. I should like to point out that many "tumors" of the soft parts of the extremities are not actually neoplasms. Such conditions as hematomas, cold abscesses, ganglion cysts, and popliteal cysts, all of which are tumors, are not actually neoplasms.

Many of these tumors arise in an insidious manner with little or no pain, but, nevertheless, they are malignant and just as destructive of life as any malignant tumor. Patients and even physicians may, at times, regard them as of no serious consequence, and much time is lost in starting adequate treatment. Oftentimes biopsy is done with no provision made for immediate definitive surgical treatment, should it be necessary.

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Section of Orthopedic Surgery, Mayo Clinic and Mayo Foundation. The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

Clinical diagnosis in most of these cases cannot be made without the aid of a pathologist, and, therefore, operative exploration is necessary. Only two of these types of tumor may be accurately diagnosed clinically without surgical exploration. Certain lipomas and some hemangiomas have characteristic roentgenographic pictures. These will be described briefly later.

For several years we of the Section of Orthopedic Surgery of the Mayo Clinic have been accumulating and studying material regarding these tumors in an effort to arrive at a better understanding of this problem. From the beginning it was necessary to seek the aid of the Section of Surgical Pathology in the study of these cases. Several groups of cases were studied by members of our Section with the advice and collaboration of the pathologists. Finally, Dr. Soule,[5] of the Section of Surgical Pathology, made a study of the records of 500 consecutive cases of soft-tissue tumors of the extremities and published his results. His tabulation of the incidence of the various tumors is shown in the Table.

Incidence of Tumors of the Soft Tissue of the Extremities

Type of Tumor	Incidence by per Cent		
	Entire Series	Malignant Disease in Each Group	Malignant Tumors Only
Fibromatous	12	67	55
Lipomatous	33	3	7
Angiomatous	13	5	4
Neuromatous	19	4	5
Myomatous	3	38	7
Synovial	9	22	14
Miscellaneous	11	11	8

The following classification of these tumors published by Soule is a modification of Stout's classification.

- Fibromatous Tumors
  - Benign
    - Keloid
    - Fibromatosis
      - Palmar
      - Plantar
    - Fibroma
    - Fibromyxoma (myxoma)
    - Extra-abdominal desmoid tumor
  - Malignant
    - Dermatofibroma protuberans
    - Low-grade fibrosarcoma
    - Fibromyxosarcoma (myxosarcoma)
    - Fibrosarcoma
- Lipomatous Tumors
  - Benign
    - Lipogranuloma
      - Traumatic cysts
      - Infectious granuloma
      - Idiopathic
    - Hibernoma
    - Lipoma
  - Malignant
    - Liposarcoma (lipomyxofibrosarcoma)
- Angiomatous Tumors
  - Benign
    - Hemangioma
      - Capillary
      - Cavernous
      - Mixed type
    - Lymphangioma
    - Glomus tumor
    - Hemangiopericytoma
  - Malignant
    - Hemangioendothelioma
    - Angiosarcoma
    - Lymphangiosarcoma
    - Malignant hemangiopericytoma
- Myomatous Tumors
  - Benign
    - Leiomyoma
    - Granular-cell myoblastoma
    - Vascular myoma
  - Malignant
    - Leiomyosarcoma
    - Malignant granular-cell myoblastoma
    - Rhabdomyosarcoma
- Neuromatous Tumors
  - Benign
    - Traumatic neuroma
    - Sclerosing neuroma
    - Neurilemmoma
    - Neurofibroma (von Recklinghausen)
  - Malignant
    - Neurofibrosarcoma (malignant schwannoma)
    - Neuro-epithelioma (peripheral ependymoma)
- Synovial Tumors
  - Benign
    - Ganglion cyst
    - Baker's cyst and adventitious bursae
    - Giant-cell tumor of tendon sheath
    - Osteochondromatosis of bursae
  - Malignant
    - Synovial-cell sarcoma
- Miscellaneous Tumors
  - Benign
    - Fibrous xanthoma
    - Mesenchymoma
    - Myositis ossificans
  - Malignant
    - Malignant mesenchymoma
    - "Alveolar cell sarcoma"
    - Reticulum-cell sarcoma and allied lymphomas

Time does not permit a detailed discussion of each of these tumors. From a practical standpoint, I have elected to discuss the following types in some detail:

- Plantar fibromatosis
- Extra-abdominal desmoid tumor
- Fibrosarcoma
- Lipomas
- Liposarcomas
- Hemangiomas
- Glomus tumors
- Rhabdomyosarcoma
- Traumatic neuroma
- Ganglion cyst
- Baker's cyst
- Giant-cell tumor of the tendon sheath
- Synovial-cell sarcoma

Selection of these particular tumors is made because I have had more experience with them than with the others mentioned in the classification, and I believe anyone interested in the general problem will have had experience with these same types.

CONSIDERATION OF SPECIFIC TUMORS

Plantar Fibromatosis.—In this condition a benign lesion involves the plantar fascia of the foot; it seems to be more prevalent now than it was some years ago. Patients are often disturbed by this condition, and physicians and pathologists may be confused. These lesions tend to recur rapidly after excision, and, because they show pronounced

cellular activity when examined microscopically, the pathologist may be led to diagnose them as malignant lesions. In a group of cases studied at the Mayo Clinic, metastasis was not observed, even though the lesion may have recurred locally in many instances.

The same pathologic process may involve the palmar fascia, and a similar pathologic process is found in keloids, Peyronie's disease, and certain other localized fibrous tissue formations that are less commonly found.

Extra-Abdominal Desmoid Tumor.—This benign unencapsulated tumor arises from the musculoaponeurotic structures and locally invades the surrounding muscle fibers. It does not metastasize but tends to recur locally and to invade other tissues locally; these tendencies make treatment a very difficult problem. The term "desmoid," meaning bandlike, was coined by Müller [6] in 1838 to describe the tendon-like appearance of such tumors, and, while the lesion was first described affecting the abdominal wall, the occurrence of the lesions in other parts of the body is now recognized.

Various theories as to the cause of these tumors have been offered. Trauma was considered a cause because of the possibility of ruptures of muscle fibers in the abdominal wall during the violent muscular contractures of labor. Endocrine origin has been suggested by Geschickter and Lewis,[7] who found 13,000 rat units of gonadotropic substance per kilogram of tumor. Other observers have supported this theory by various observations.

The incidence of this tumor in Soule's study was 11% of the fibromatous group. Young and McDonald [8] collected 50 cases from the records of the Mayo Clinic. Characteristically, the tumor appears as a mass of fibromatous tissue that invades and surrounds muscle fibers. The most difficult differential diagnosis arises from slowly growing fibrosarcomas.

The accepted treatment of this tumor is early and wide surgical excision. The excision must be wide or recurrence will take place. In some tumors involving extremities, the invasion and surrounding of blood vessels and nerves may make amputation necessary in order to effect a cure.

Fibrosarcomas.—Fibrosarcomas make up the largest and probably the most important group of the fibromatous tumors. In Stout's [1] monograph the tabulation of 1349 malignant mesenchymal tumors from the surgical pathology laboratory of Columbia University shows fibrosarcoma to be by far the most frequently seen tumor. In Soule's series 55% of the malignant tumors were fibrosarcomas.

Fibrosarcomas are tumors of connective-tissue origin and may arise from fascial, subcutaneous, or fibrous intermuscular tissue as well as from intramuscular septa. They often appear to be encapsulated; this appearance may be deceiving, for this "capsule" may be composed of neoplastic cells that show active growth. These tumors may be graded according to the degree of malignancy.

Treatment should be surgical. Wide excision may be sufficient in low-grade tumors, provided the excision is well beyond all possible limits of the tumor. However, it is our conviction, based on our experience to date, that many more patients having these tumors will be saved by primary amputation than by complete eradication by means of local excision, if the outcome of excision is at all doubtful. Many patients will not consent to primary amputation, and the surgeon must resort to local excision. After this fails, consent to amputation may be gained. This, however, is often too late.

In 107 cases studied by Ivins [9] more than 55% of the patients died of fibrosarcoma. The patients with low-grade tumors lived longer than those with high-grade tumors. The percentages of patients who died were as follows: 28% having tumors of Grade 1; 54% with tumors of Grade 2; 64% with tumors of Grade 3, and 71% with tumors of Grade 4.

Lipomas.—These tumors comprise a third of the entire group of soft-tissue tumors of the extremities reported by Soule.[5] Most

lipomas occur in the fatty subcutaneous tissues and are usually readily recognized by the experienced physician on simple palpation and inspection. As a rule, they are relatively small and often lie dormant for many years, with no signs of growth and no painful symptoms. If lipomas become large, and particularly if they are situated beneath the fascia, in intermuscular septa, and around the capsules of the larger joints, a characteristic roentgenographic picture is presented. This is manifested by an outline of denser shadow, representing the capsule surrounding the tumor, and by a clearer radiolucent portion extending through the tumor. Lipomas are best treated by surgical removal. Care must be taken to see that the tumor is removed entirely, or recurrence may take place. Any lipoma which shows signs of active growth or which becomes painful should be excised.

Liposarcoma.—Kimbrough [10] found 23 cases of liposarcoma of the extremities in the records of the Mayo Clinic for the years 1915 through 1951. All but one of the liposarcomas occurred in the lower extremities. Thirteen were in the thigh, three in the popliteal space, four in the leg, and two in the gluteal region. In Stout's tabulation of the cases from the pathologic laboratory of Columbia University, liposarcomas were second in frequency among the malignant mesenchymal tumors. It should be pointed out that Stout's group included tumors from all parts of the body, whereas Kimbrough's group included tumors from the extremities only.

The tumor is usually lobular, firm, and rubbery, although it may be soft and gelatinous. Stout has outlined liposarcomas according to cellular differentiation as differentiated and undifferentiated liposarcomas. It is Stout's opinion that the great majority of liposarcomas do not arise from preexisting lipomas. His second group may show "less differentiated fat" and may have "myxoid areas with extremely bizarre giant cells often with huge pyknotic nuclei."

Regarding treatment Stout stated, "Since only small superficial tumors 4 cm. or less in diameter have proved curable by radiotherapy, excision or amputation is the treatment generally selected." Pack [11] stated, "Although not always possible of achievement, a good rule to follow is to remove the liposarcoma without seeing or encountering it. . . . In cases of amputation for liposarcomas the upper level of severance must be high if not extreme sometimes fore or hind quarter amputations. The general rule of amputating above the level of origin of the muscle groups involved by the sarcomas is a good principle to observe."

Kimbrough's conclusions [10] from his study of the cases at the Mayo Clinic were as follows: "Analysis of the use of x-ray therapy in this series does not indicate that liposarcoma can safely be considered to respond well to irradiation therapy. Surgical excision is a temporary approach to the treatment of these tumors, but can be expected to be followed by recurrence in 61 per cent within 14 or 15 months, and two thirds of these are likely to have distant metastases.

"Amputation seems to be the most favorable surgical approach. There were no recurrences following amputation in this series in spite of previous recurrences after local excision."

Hemangiomas.—A large part of the group of vascular tumors is made up of hemangiomas. These arise from that portion of the mesenchyme which forms blood and lymph vessels. The congenital origin of these tumors is widely recognized and accepted. Some may be discrete and others diffuse. Johnson [12] analyzed hemangiomas in 93 cases at the Mayo Clinic and found 20 diffuse and 73 discrete. Pain and presence of a mass of tissue are the commonest symptoms.

Roentgenographic changes may be noted, such as soft-tissue tumor, periosteal thickening or bony erosion where the mass is adjacent to the bone, and phleboliths. Surgical excision is the treatment of choice and in discrete hemangiomas may be expected to be followed by cure. In the diffuse types a much more difficult problem is encountered; complete removal often is difficult or impossible,

and symptoms of varying degree usually persist.

Glomus Tumors.—These tumors are small, benign, and very painful and are usually found in the subcutaneous tissues of the extremities. There were eight glomus tumors among the 500 tumors studied by Soule. Grossly, these tumors are deep red, blue, or purple and vary greatly in size, ranging from the size of a pinhead to that of a walnut.

Pain is the outstanding symptom and tenderness the outstanding sign. The patient usually will respond with violent withdrawal on palpation of the tumor. At times, the patient may not permit the examining physician to touch the tumor. Discoloration of the skin or nail in the more superficial tumors may be noted. Excision is the treatment of choice and is followed by cure.

Rhabdomyosarcomas. — These malignant neoplasms arise from striated muscle fibers, although it is not definitely known whether all of them are connected with striated voluntary muscle. In our experience they are relatively uncommon, there being only three cases in Soule's series of 500 tumors of soft tissues of the extremities. However, Pack and Eberhart [13] stated that rhabdomyosarcomas represent about 20% of the soft-tissue sarcomas.

Commonly these tumors are not painful until they have become very large. The result is that they are usually discovered accidentally and may have reached a fair size before being discovered. Treatment should be by radical excision or, preferably, by amputation when possible.

Traumatic Neuromas.—Strictly speaking, traumatic neuromas are not neoplasms, but, rather, they represent a reparative process in nerves subjected to trauma. From the orthopedic standpoint the most important of these tumors is the plantar neuroma. Of Soule's 500 cases, 23 were plantar neuromas. They occur more frequently in women than in men. They are found along the plantar digital nerves, usually to the third and fourth toes, and develop from repeated traumatism to that nerve.

The main symptom is a sharp pain in the involved toe; often the pain is so severe as to cause the patient to stop walking or standing and take off his shoe and massage and manipulate the foot until relief is obtained. On physical examination a very painful spot usually presents on the plantar surface of the foot beneath the site of the tumor. Pressure on this spot may reproduce the severe pain along the toe.

Relief is obtained by surgical excision of the neuroma.

Ganglion Cysts.—Such cysts are well-known tumors involving the articular structures or tendon sheaths and are often found about the wrist joint or the foot and ankle joint. Contrary to widespread belief, ganglions usually arise from the joint proper passing by a tendon sheath and often adherent to it. Gradual enlargement takes place as the ganglion becomes filled with a clear mucinous substance of varying viscosity. These tumors are benign, and pain varies in degree; often they are more unsightly than painful.

Treatment of various sorts has been advocated, such as forcible rupture, aspiration and injection of sclerosing fluids, and excision. Careful and complete excision is, in our experience, the most satisfactory method of treatment.

Baker's Cysts.—These cysts, the counterpart of ganglion cysts, occur in the popliteal space among persons of any age. They are often painless but may slightly restrict motion in the joint if they become large. Occasionally they are seen as an extension of rheumatoid arthritis in the knee, but more frequently they are isolated synovial cysts with no involvement of the knee joint. They are fairly firm in consistency, feel somewhat elastic, and often are not tender. Occasionally they may disappear spontaneously, but oftener they persist and enlarge slowly unless removed.

Surgical excision is the treatment of choice. Careful dissection will, as a rule, show the cyst arising from the knee joint, usually medial to the inner hamstring muscles. There may or may not be an open connection with

the joint. Recurrence may occasionally take place, but malignant changes never take place.

Synovial-Cell Sarcoma.—This type of sarcoma has become more widely recognized during the past two decades. Its serious nature is now well known to everyone interested in these tumors, although its exact origin still is not clear. The term "synovial-cell sarcoma" would indicate that it originates from synovial cells, but this cannot always be demonstrated. It is one of the more frequently found malignant tumors of the somatic soft tissues. In Soule's study it was second in importance to the fibrosarcomas.

It is a neoplasm of young people, 60% of the patients being less than 30 years of age. It is the commonest soft-tissue sarcoma of the hands and feet. As a rule, it is painless.

It usually spreads by the blood stream, though regional lymphatic vessels and nodes are found to be invaded in from 10% to 15% of cases.

Once the diagnosis is definitely established, it is our opinion that radical surgical treatment is indicated, and by that I mean that amputation is usually indicated when an extremity is involved. A very pessimistic outlook on the prognosis in these cases has developed, but it is our feeling that with earlier recognition and more radical treatment a much greater percentage of 5-year survivals will be observed.

Xanthomas.—This lesion that has been given several names by various authors and that is also known as "giant-cell tumor of tendon sheath" is a lesion of variable size which may occur either in or along tendon sheaths or in joints. When joints are involved, it may be either by a discrete xanthoma or by diffuse xanthomatosis, now recognized as "pigmented villonodular synovitis."

The cause of xanthomas is not known. Changes may occur in the metabolism of lipids. The tumors are benign but may recur locally if not completely removed. The commonest location of xanthomas is along the tendons and tendon sheaths of fingers. As xanthomas enlarge they may, at times, cause erosion of bone from pressure. In rare cases xanthomas may occur in multiple locations. At times, in joints, they may reach fairly large size. In cases of pigmented villonodular synovitis, a marked enlargement of the joint may occur, particularly in cases of the knee joint.

All types of xanthomas should be surgically excised. X-ray therapy may be used as an accessory treatment.

SUMMARY

A classification of tumors of the soft parts of the extremities exclusive of the skin and lymph nodes, prepared by Soule and a modification of Stout's classification, has been presented together with brief discussions of some of the commoner tumors of this group.

It has been emphasized that in most of these tumors diagnosis without the aid of a competent surgical pathologist is not possible. It should also be emphasized that many of these tumors may be cured by earlier and more radical surgical treatment. The surgeon who undertakes an operation to establish the diagnosis in one of these cases should be prepared to go ahead with definitive treatment at once. By such a plan it is to be hoped that a much higher percentage of 5-year survivals will be obtained.

DISCUSSION

DR. HERBERT M. NICHOLS, Portland, Ore: My experience with these tumors, while fairly extensive numerically, is not large enough to be of any statistical value. Since most of my cases are confined to the upper extremity, the distribution is not quite the same as it would be for both upper and lower extremities.

Among the benign tumors, I have been faced with various types of fibromas, xanthomas, hemangiomas, lymphangiomas, and of course lipomas and numerous ganglions. Malignant tumors which only constitute 10% of hand tumors have not been at all common. Tumors which derive from peripheral nerve elements have been fairly common. I have three interesting cases which I should like to show very briefly.

CASE 1.—A white man, age 50. History of painless swelling in the distal palm of the right hand of three or four months' duration [Slide]. Examination showed a soft, ill-defined tumor which lay in the

web between the long and ring fingers with a prolongation extending back along the fourth metacarpal. At operation the tumor tissue appeared to be composed of numerous small cysts filled with jelly-like material which infiltrated all the tissues along the nerves and artery of the third cleft, lying beneath the palmar fascia and extending back into the carpal canal. Surgery was carried out in February, 1952. Although the tumor was classified as a myxoma [Slide] by the pathologist, it has not recurred.

CASE 2.—This white woman 68 years old gave a history of a tumor of the left forearm of 14 years' duration. She had had five operations with recurrences. The tumor [Slide] involved the dorsum and ulnar border of the lower forearm with fixation to tendon and bone, and the extremity was painful and useless. She refused amputation. Surgery consisted of radical excision of all involved tissue with application of abdominal pedicle to fill the defect. Surgery was carried out in 1947. I saw her last in 1954, at which time she was perfectly well and there were no signs of recurrence. The pathology in this case [Slide] was a neurofibroma.

CASE 3.—This 25-year-old white man first had a neurofibroma removed from the left side of his neck when he was 8 years old. I first saw him 8 years ago when he was 16. He had a huge plexiform neurofibroma 30 cm. by 18 cm. of the left side of the neck. I removed as much of it as possible surgically and then had him treated by x-ray. He remained well again for eight years then returned with complaints of hoarseness, coughing, and vomiting. A tumor of the thoracic inlet 10 cm. long [Slide] was found and removed. This was a malignant schwannoma [Slide]. Further studies showed the presence of a mediastinal tumor of the vagus nerve [Slide]. This was removed (by Dr. John F. Higginson) and found to be benign. He is now developing pigmentary changes and other neurofibromas [Slide]. The final diagnosis was von Recklinghausen's disease.

The sequence of events of this last case might have occurred in an extremity. Amputation in any of these three cases, while seemingly indicated, would not have been necessary.

DR. RALPH K. GHORMLEY, Rochester, Minn.: I believe that Dr. Nichols' discussion emphasizes the point that accurate diagnosis of these tumors depends on the aid of a good pathologist. I remember talking on this subject with a very prominent surgeon who is interested in this problem, and he stated that one of the greatest troubles is to get the pathologists to agree on a diagnosis of these tumors.

Many of these tumors are not common and are not often seen. One should really never amputate for a benign tumor. Patients may not consent to amputation even if it is diagnosed as malignant, but, when you are sure of a malignant tumor and amputation is the best treatment, the patient should be so informed.

REFERENCES

1. Stout, A. P.: Tumors of the Soft Tissues, Sect. II, Fascicle 5, Washintgon, D. C., Armed Forces Institute of Pathology, 1953, 138 pp.

2. Pack, G. T.: End Results in the Treatment of Sarcomata of the Soft Somatic Tissues, J. Bone & Joint Surg. **36-A**:241 (April) 1954.

3. Cade, S.: Soft Tissue Tumours: Their Natural History and Treatment, Proc. Roy. Soc. Med. **44**:19, 1951.

4. Jönsson, G.: Malignant Tumors of the Skeletal Muscles, Fasciae, Joint Capsules, Tendon Sheaths and Serous Bursae, Acta radiol. Suppl. **36**:7, 1938.

5. Soule, E. H.: Primary Soft-Tissue Tumors of the Extremities: Classification, Histogenesis and Incidence, American Academy of Orthopedic Surgeons, Instructional Course Lectures **11**:3, 1954.

6. Müller, J.: Über den feiner Bau und die Formen der krankhaften Geschwülste, Berlin, G. Reimer, 1838, 60 pp.

7. Geschickter, C. F., and Lewis, D.: Tumors of Connective Tissue, Am. J. Cancer **25**:630 (Nov.) 1935.

8. Young, H. H., and McDonald, J. R.: Extra-Abdominal Desmoid Tumors, American Academy of Orthopedic Surgeons, Instructional Course Lectures **11**:14, 1954.

9. Ivins, J. C.: Fibrosarcoma of the Soft Tissues of the Extremities, American Academy of Orthopedic Surgeons, Instructional Course Lectures **11**: 18, 1954.

10. Kimbrough, R. F.: Liposarcoma of the Extremities, Thesis, Graduate School, University of Minnesota, 1951.

11. Pack, G. T., and Pierson, J. C.: Liposarcoma: A Study of 105 Cases, Surgery **36**:687 (Oct.) 1954.

12. Johnson, E. W., Jr.: Hemangiomas of the Extremities, American Academy of Orthopedic Surgeons, Instructional Course Lectures **11**:28, 1954.

13. Pack, G. T., and Eberhart, W. F.: Rhabdomyosarcoma of Skeletal Muscle: Report of 100 Cases, Surgery **32**:1023 (Dec.) 1952.

Axillary Cystic Hygroma

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and
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Cystic hygroma of the axilla is a rare tumor. Dowd [1] in 1913 was able to collect only 35 cases from the world literature. Watson and McCarthy [2] reported 14 cases of hygroma, only 1 of which was axillary in position, in a series of 1056 cases of blood- and lymph-vessel tumors seen at the New York Memorial Hospital over a nine-year period. The first case of cystic hygroma was reported in 1828, and the condition has been more widely recognized since Wernher's [3] publication in 1843. Since that time, isolated cases have been reported in detail or the lesion has been briefly referred to in papers dealing with the commoner neighbor lesion, cystic hygroma of the neck. Dowd [1] indicated the relative frequency of cystic hygroma of the neck to that of the axilla to be 91 to 35 in his collected series. Gross [4] in his personal series of 112 cases encountered 2 in the axilla, 101 in the neck, and 9 in miscellaneous sites.

ETIOLOGY

The etiology of cystic hygroma has been a subject of much interest. Wernher [3] first described the lesion as a new growth, but it was not until Sabin's [5] work on the development of the lymphatic system that real progress was possible. McClure and Silvester [6] had previously suggested that hygroma arose from sequestrations of lymphatic tissue derived from the primative jugular sacs, and Goetsch [7] was able to offer support to this theory and clarify the manner in which growth of the lesion occurs. He demonstrated that endothelial sprouts from the walls of marginal cysts penetrate adjacent normal tissues and through secretion become canalized and form enlarging cysts. Finally these cysts, engulfing and exerting pressure upon normal structures, cause their destruction.

PATHOLOGY

Goetsch [7] has recorded the most complete pathological description of cystic hygroma. It is a benign, true neoplastic tumor of lymphatic origin. It is a multilobular, multilocular cystic tumor, the cavities of which are lined by endothelium. The walls of young cysts are thin, delicate, and practically transparent, whereas those of older cysts are often thick and fibrotic. Lymphoid tissue and even germinal centers are frequent in the interlobular stroma, which also exhibits the characteristic inflammatory reaction in response to infection. The fluid content is usually clear or straw-colored but occasionally turbid or blood-stained. It is practically free of albumin and globulin, and various types of leucocytes are present.

REPORT OF CASES

This presentation is concerned with two patients with axillary cystic hygroma admitted to the University of Alberta Hospital within a three-day period, both of which underwent total surgical removal with satisfactory results.

Our first case is that of a 16-month-old girl admitted on Dec. 23, 1954. The only significant past history was of an attack of whooping cough five weeks previously. The mother stated that the child had a mass under the left arm, which had appeared five months previously but which had enlarged in an alarming fashion in the three weeks immediately preceding admission.

Figure 1 is a preoperative photograph.

On examination the significant findings were limited to the left axilla. In this situation a well-circumscribed, smooth, multilobular, nontender tumor mass was apparent, extending from the posterior axillary line to within 3 cm. of the sternum and superiorly from the apex of the axilla to a point 10 cm. (4 in.) below this. The mass on trans-

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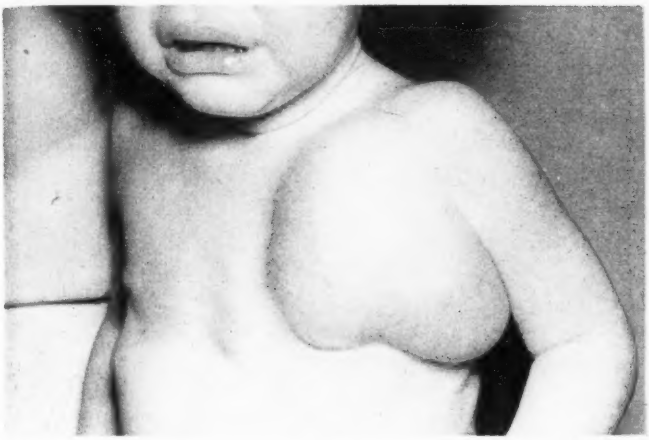

Fig. 1 (Case 1).—Preoperative photograph.

illumination was not attached to skin, and distended superficial veins coursed over its surface. The clinical diagnosis was axillary cystic hygroma.

Admission laboratory investigation was normal except for the x-ray of the chest (Fig. 2). This showed a spherical soft-tissue swelling approximately 4 in. in diameter arising from the left lateral chest wall and extending into the pectoral and axillary regions. It was of homogeneous density, and the appearance was considered to be consistent with a diagnosis of benign cystic tumor.

Operation was performed with ether anesthesia on Dec. 31. The incision began at the midpoint of the clavicle, passed inferiorly, and then swept posteriorly over the most prominent portion of the tumor to the posterior axillar line. Development of skin flaps demonstrated the tumor to pass deep to the attenuated latissimus dorsi and pectoralis major muscles, both of which it invaded, as it did the pectoralis minor. The inferior part of the dissection was relatively simple, though excision of portions of the muscles referred to was necessary to insure total removal. Superiorly, the proximity of the mass to the axillary vessels and brachial plexus made identification of these structures necessary before the final pedicle of the lesion was isolated. Section of this pedicle, which terminated adjacent to the axillary vein, permitted removal of the lesion intact. The wound was closed with drainage.

Figure 3 is a photograph of the sectioned gross specimen and illustrates well its multilobular structure and the glistening endothelial lining of the cysts. Figure 4 is a representative histological section and shows the endothelial-lined cysts and loose stroma.

The child's postoperative course was complicated by a bronchopneumonia which responded rapidly to chloramphenicol (Chloromycetin), and she was discharged 15 days after operation. Follow-up reports from the family physician indicate the child

Fig. 2 (Case 1).—Chest x-ray.

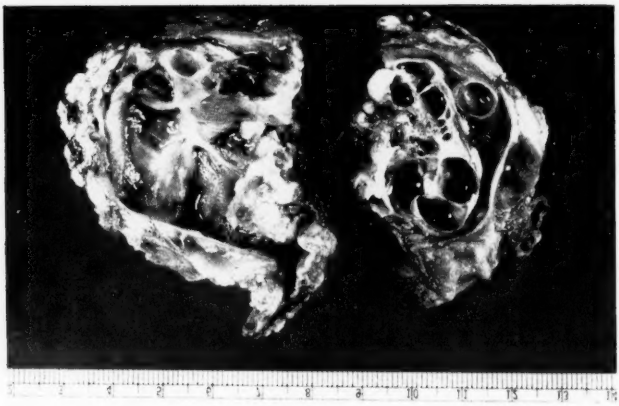

Fig. 3 (Case 1).—Sectioned gross specimen.

to be well and without evidence of recurrence at the time of reporting, 11 months postoperatively.

Our second case is that of a 4-year-old boy admitted on Dec. 26, 1954, as an emergency. The history was one of spontaneous development of pain in the right arm pit 48 hours prior to admission in a previously healthy child. At this time a mass was apparent in this situation, which enlarged with alarming rapidity. There was no history of trauma or recent infection.

The temperature was 101.4 F, but otherwise the significant findings on examination were limited to the right axilla as illustrated in Figure 5. In this situation a tumor mass 9 cm. in diameter was present. It extended from the clavicle superiorly, anteriomedially deep to the pectoralis major muscle to within 3 cm. of the midsternal line, and posteriorly elevating the latissimus dorsi muscle and the scapula off the chest wall. The mass was diffuse, without well-defined borders, was soft, though not definitely fluctuant, and gave the impression of being lobulated. It did not transilluminate and though moderately tender did not exhibit local heat or redness. A few engorged veins were present over the mass. The clinical diagnosis was hematoma.

The laboratory investigation was completely normal, save for a leucocyte count of 13,500, of which 76% were polymorphonuclear, and a chest x-ray (Fig. 6) which was essentially similar to that previously reported, save for the soft-tissue lesion being on the opposite side.

Three days after admission, the mass was aspirated in the operating room and 30 cc. of clear pale-yellow fluid was obtained. This proved to be sterile on culture and had a protein content of 5.46 gm. per 100 cc., of which the albumin fraction was 3.45 gm. and the globulin 2.01 gm. The fine clot that formed on standing was composed of fibrin and lymphocytes, the sediment contained lymphocytes and polymorphonuclear neutrophiles.

The diagnosis was now considered to be cystic hygroma, and operation was carried out with the patient under ether anesthesia on Jan. 5, 1955. The incision was the same as that used in the first case,

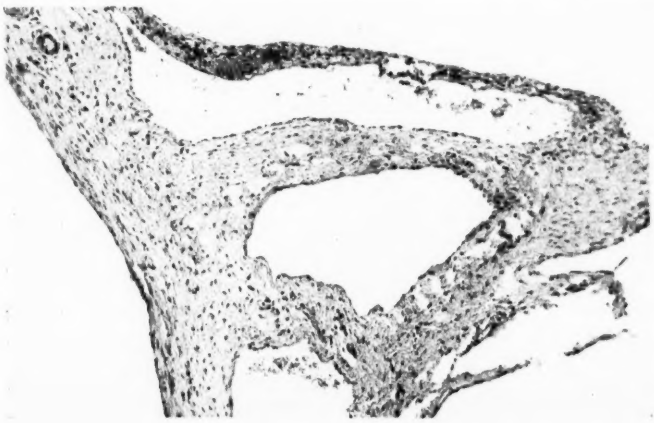

Fig. 4 (Case 1).—Microscopic section of specimen.

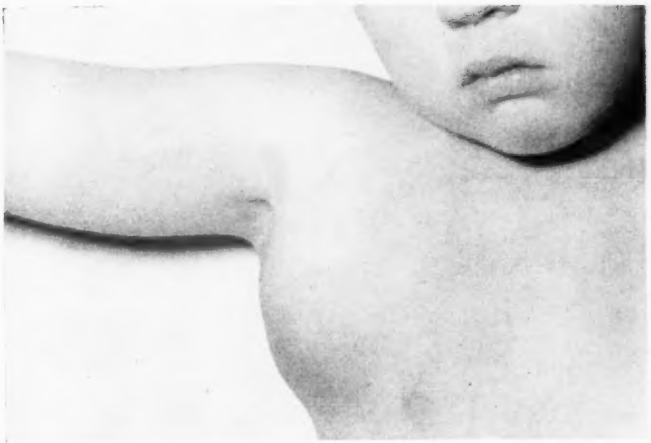

Fig. 5 (Case 2).—Preoperative photograph.

and the operative findings were almost identical as far as the inferior part of the dissection was concerned, save that the nerve to latissimus dorsi coursed over the superficial aspect of the tumor. Superiorly, the tumor mass narrowed to a broad pedicle which passed posterior to the axillary vessels and brachial plexus and disappeared toward the apex of the axilla. In addition, the pedicle was traversed by the circumflex nerve, preservation of which made opening of one of the large cystic spaces necessary. Traction was then exerted on the pedicle, and it was sectioned as high up as possible. The wound was closed with drainage.

Figure 7 is a representative microscopic section of the lesion and shows the same basic pattern as was seen in the first case.

The child's postoperative course was remarkably uneventful, and he was discharged 13 days after operation. Follow-up at the time of reporting with an 11-month interval indicates the child to be well and free of recurrence.

TREATMENT

Over the years many methods of treating these lesions have been in vogue, including expectant treatment, simple aspiration, aspiration and injection of sclerosing agents, radium and x-ray irradiation, and surgical excision. Recent authors appear to be in agreement that surgical excision in the case of cervical * and axillary † cystic hygromas is the treatment of choice and carries an acceptable morbidity and mortality. Ward,

* References 4, 9, and 10.

† References 10, 11, and 12.

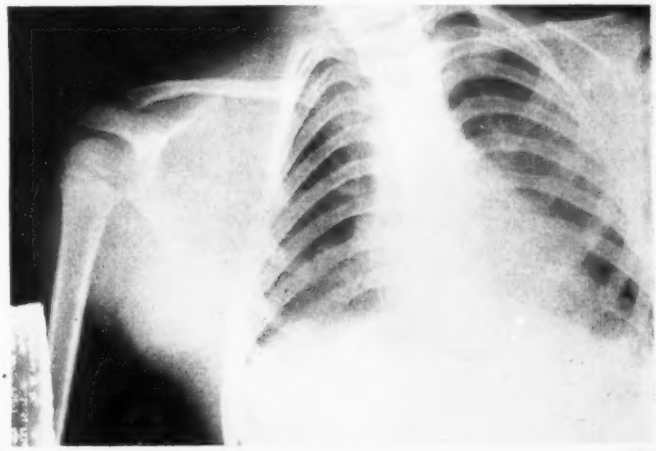

Fig. 6 (Case 2).—Chest x-ray.

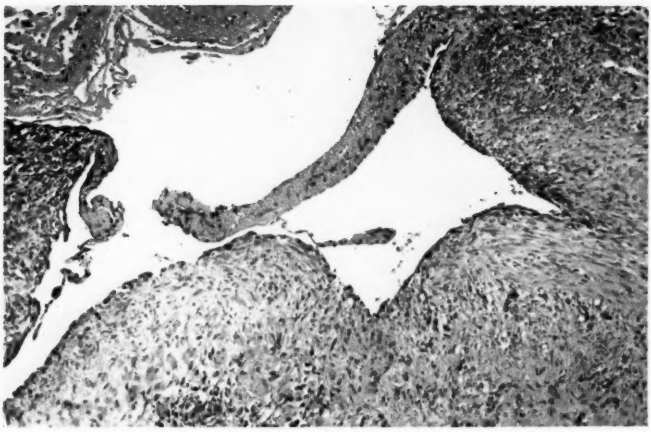

Fig. 7 (Case 2).—Microscopic section of specimen.

Hendrick, and Chambers [13] recommend the use of preoperative sclerosants, but this practice does not appear to be widely accepted.

COMMENT AND CONCLUSION

The rapid development of the hygroma in our second case with the resulting erroneous admission diagnosis has been very interesting to us, and we were of the opinion that such an occurrence must be unique. Whereas such a manner of presentation is certainly uncommon, an almost identical case was reported by Heer.[8] We must therefore entertain the diagnosis of cystic hygroma in axillary masses of acute onset as well as those persuing a more chronic course.

Our own limited experience would indicate that surgical excision is a very satisfactory method of dealing with cystic hygroma as it occurs in the axilla.

DISCUSSION

Dr. William B. Hutchinson, Seattle: We are certainly indebted to Drs. Macbeth and MacKenzie for presenting these two very unusual and rare cases.

To give you some idea of how rare they are, at the Swedish Hospital in the last 10 years 37,000 major operations have been done and not one single cystic hygroma of the axilla has been encountered. At the Seattle Children's Orthopedic Hospital in the last five years a little more than 6500 major operations have been done, and one case of cystic hygroma of the axilla was encountered.

I should like to ask Dr. Macbeth if in the second case he presented he felt that the history of trauma was accurate. Actually it is not unusual, as he mentioned, to see these cystic hygromas present a swelling. The usual place, of course, is in the neck. These may occur very rapidly. This is due perhaps to the fact that the lymphangiomatous mass, which is there originally, sends out endothelial buds or cysts and presents this swelling.

Anyone who has tried to get a cystic hygroma out of an area where there are vital structures will realize the magnitude of the task. This is unquestionably due to what the essayists emphasized—the endothelial sprouts from the walls of marginal cysts penetrating adjacent normal tissues and forming enlarging cysts. The resulting pressure of these cysts that encircle normal structures eventually causes their destruction. When one begins to take out this mass, especially when there are vital structures, it certainly is extremely difficult.

I should like to thank Dr. Macbeth for at least calling to my attention the clinical entity of cystic hygroma of the axilla.

Dr. Kenneth C. Sawyer, Denver: This is a timely and most interesting paper and has been very well presented. I should also like to emphasize the importance of early surgical excision of all cystic hygromas.

[Slide] This tumor in a 6-week-old child was easily excised, although it extended into the mediastinum and deep to the great vessels. [Slide] The uncollapsed portion of the gross specimen is shown here. [Slide] The thin-walled, nonadherent tumor is microscopically depicted in this slide. The lack of infiltration into the neighboring tissues probably accounted for the easy dissection.

[Slide] This is a photograph of a 10-month-old boy with a history of a tumor in the neck that was noted three weeks prior to admission. Its removal was accomplished with little difficulty. [Slide] This microscopic section, however, shows considerably

more fibrosis than the section in the 6-week-old infant.

[Slide] The tumor shown in this 3-year-old child was much more adherent to the surrounding structures and was very difficult to remove because a sclerosing agent had been injected into the tumor. [Slide] The inflammatory process in the cyst wall can be seen in this photomicrograph.

[Slide] The hygroma you see here was removed from a 33-year-old man. It presented quite a technical problem because of the dense fibrosis of its walls —[Slide] as illustrated in this slide.

At Children's Hospital we are convinced (and we become more convinced with each case) that, regardless of the extent of any hygroma, its removal is less complicated if it is attempted when first seen.

DR. CARL B. DAVIS JR., Chicago: I do not want to prolong this discussion indefinitely but I should like to present to you an interesting case—interesting from two standpoints: first, because we saw this boy initially 10 years ago; second, because of the rarity of the location of this cystic lymphangioma.

[Slide] This shows the location of the large tumor of the upper thigh and leg found in this infant at 4 months of age. The mother stated it was noticed that the mass increased in size and then again regressed. There was no history of trauma. The lesion was excised in one operative procedure through these two incisions. An extensive dissection was undertaken.

Approximately 10 years later, residual tumor appeared near the patella. The child was operated upon again, and the same type of tumor found as observed at the first operation.

[Slide] This shows you essentially the same pathology demonstrated in the other cases, namely, the endothelial lining of the cystic spaces which contained clear watery fluid.

[Slide] This shows the histology of the specimen removed at the second operation (10 years after the first operation), showing the dense scar tissue in which there are these dilated lymphatic-like spaces.

This patient was seen a short time ago. At that time there was no evidence of any demonstrable tumor.

DR. ROBERT A. MACBETH, Edmonton, Alta., Canada: First, I should like to thank the Association for the privilege of appearing before this meeting. I should also like to thank the discussers, Drs. Hutchinson, Baker, Sawyer, and Davis. I will try to answer the questions they raised.

First of all, there was no history of trauma in the second case, in which the tumor developed so quickly. We initially diagnosed the lesion as hematoma and therefore inquired particularly in this regard, but as far as we were able to determine it developed spontaneously.

As far as the timing of the procedure is concerned, no one person has any great experience with this particular lesion, but I believe it is generally conceded (and certainly it is our own feeling) that the operation should be done when the condition is first recognized and presented for treatment.

As far as staging is concerned, it was not necessary in either of our cases, since they were limited to one anatomic region. However, there are reports in the literature of cases involving the neck and mediastinum, neck and axilla, etc., where the staged procedure has apparently been carried out quite successfully.

REFERENCES

1. Dowd, C. N.: Hygroma Cysticum Colli: Its Structure and Etiology, Ann. Surg. **58**:112-132 (July) 1913.

2. Watson, W. L., and McCarthy, W. D.: Blood and Lymph Vessel Tumors: A Report of 1056 Cases, Surg. Gynec. & Obst. **71**:569-588 (Nov.) 1940.

3. Wernher, A.: Die angeboren Kystenhygrome und die ihnen verwandten Geschwülste in anatomischer, diagnostischer und therapeutischer Beziehung, Giessen, G. F. Heyer, Vater, 1843, p. 76.

4. Gross, R. E.: The Surgery of Infancy and Childhood: Its Principles and Techniques, W. B. Saunders Company, Philadelphia and London, 1953, p. 960.

5. Sabin, F. R.: Development of the Lymphatic System in Keibel, F., and Mall, F. P.: Manual of Human Embryology, J. B. Lippincott Company, Philadelphia and London, 1912, Vol. 2, pp. 709-745.

6. McClure, C. F. W., and Silvester, C. F.: A Comparative Study of the Lymphatico-Venous Communications in Adult Mammals, Anat. Rec. **3**:534-551 (Oct.) 1909.

7. Goetsch, E.: Hygroma Colli Cysticum and Hygroma Axillare: Pathologic and Clinical Study and Report of 12 Cases, Arch. Surg. **36**:394-479 (March) 1938.

8. Heer, W.: A Case of Cystic Lymphangioma of the Axilla and of the Infraclavicular Region, Schweiz. med. Wchnschr. **61**:572 (June 13) 1931.

9. Sawyer, K. C., and Woodruff, R.: Cystic Hygromas of the Neck, A. M. A. Arch. Surg. **63**:83-91 (July) 1951.

10. Freeman, G. C.: Conservative Surgical Treatment of Massive Cystic Lymphangioma with a Report of 8 Cases, Ann. Surg. **137**:12-17 (Jan.) 1953.

11. Rankine, J. A.: Cystic Lymphangioma of the Axilla, Canad. M. A. J. **67**:667-669 (Dec.) 1952.

12. Madding, G. F., and Hershberger, L. R.: Cystic Hygroma of the Axilla: A Case Report, Am. Surgeon **20**:1003-1006 (Sept.) 1954.

13. Ward, G. E.; Hendrick, J. W., and Chambers, R. G.: Cystic Hygroma of the Neck, West. J. Surg. **58**:41-47 (Feb.) 1950.

Hyperparathyroidism, with Particular Reference to Treatment

Review of Two Hundred Seven Proved Cases

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and
JAMES F. ZIMMER, M.D., Rochester, Minn.

The treatment of hyperparathyroidism has been discussed in considerable detail previously. Practically all the principles on which treatment is based were first set forth in a remarkably prescient paper by Churchill and Cope [1] in 1934. The surgical aspects were again discussed by Cope [2] in 1941 and by Rienhoff [3] in 1950. The experience at the Mayo Clinic, based on the treatment of 63 patients, was reviewed in 1948 [4] and again after treatment of 112 patients, in 1953.[5] Another review of the details of treatment does not seem warranted at this time, but some general impressions can well be considered. The increased experience of the present has provided a better understanding of the pathology of hyperparathyroidism and a far different conception of the anatomy of the parathyroids and of the frequency of mediastinal adenomas. Somewhat surprisingly, the surgical difficulties stressed in practically all previous papers have proved far from insurmountable. In fact, the treatment of hyperparathyroidism, as judged by the experience at the Clinic, is more certain and satisfactory than that of any other surgical endocrinopathy, even including that of diseases of the thyroid.

Read at the 63d Annual Meeting of the Western Surgical Association, Seattle, Dec. 1, 1955.

Section of Surgery, Mayo Clinic and Mayo Foundation (Dr. Black), Fellow in Surgery, Mayo Foundation (Dr. Zimmer). The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

NATURE OF PRESENT SERIES

Through 1954, two hundred seven cases of hyperparathyroidism have been encountered at the Mayo Clinic in which the diagnosis has been proved at pathologic examination of tissue removed at necropsy or operation. The first surgical exploration for hyperparathyroidism at the Clinic was carried out in December, 1928, shortly after the first proved case in the United States had been encountered by Barr, Bulger, and Dixon.[6] From that time until 1942, the diagnosis was established in only 14 cases at the Clinic, in practically all of which there was advanced osseous disease. In 1942, a concerted effort was made at the Clinic to screen patients with urinary lithiasis for hyperparathyroidism,[7] as had been so successfully done by Albright and his colleagues,[8] in Boston. The effort was rewarded by the finding of four cases in the year. In the 13 years from 1942 through 1954, the diagnosis has been proved in 193 additional cases. It is still not possible to state the incidence of the condition. It is obviously not uncommon, and we see no reason to doubt the often-quoted figure of an incidence of 5% among cases of urinary lithiasis.

It is probably redundant to point out again that the condition is recognized usually because of complications involving the urinary tract or skeleton. The importance of complications in the urinary tract is shown strikingly in the series (Table 1). Renal complications, either stones or renal calcinosis, had developed in more than 80% of cases, whereas osseous complications only were found in 12% of cases. Complications of both types were present in 17% of cases. The diagnosis, as indicated in the Table, is occasionally made in the absence of such compli-

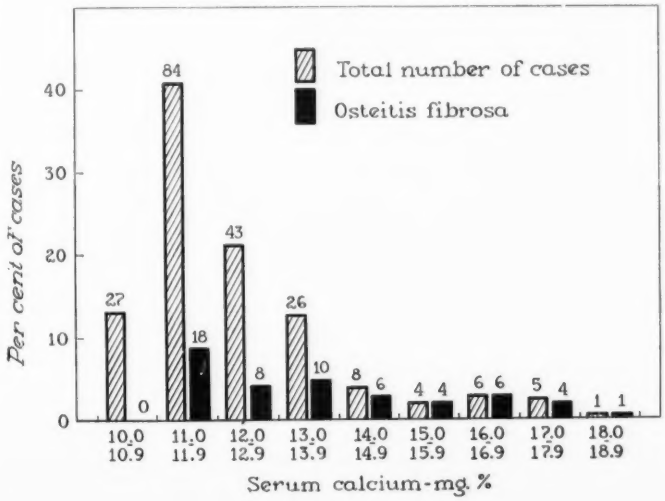
TABLE 1.—*Incidence of Complications in Proved Cases, 1928-1954*

Complications of Hyperparathyroidism	Cases	Per Cent	
Osseous only	25	12	
Osseous and urinary	35	17	82
Urinary	135	65	
Neither osseous nor urinary	12	6	
Backache	3		
Hyperinsulinism	4		
Adenoma of parathyroid found during thyroidectomy	2		
Tumor of mandible	1		
Adenoma of parathyroid found at necropsy	1		
Aching of muscles	1		
Total	207	100	

cations. Hyperfunctioning adenomas of the parathyroids were found at the time of thyroidectomy in two cases and at necropsy in one case. In four cases the question of hyperparathyroidism was raised because of the presence of hyperinsulinism. In the remaining five cases calcium determinations were carried out because of the symptoms or findings listed in Table 1. In 36 of the 207 cases peptic ulcers were present or gastric operations had been performed, presumably for ulcer. Efforts to find hyperparathyroidism among patients with peptic ulceration have been unrewarding.

The present series of cases of hyperparathyroidism differs in no way from the smaller series previously reported with respect to the sex and age of the patients and the duration of symptoms. Women outnumbered men in a ratio of 117 to 90. The age at the time of treatment varied from 14 to 74 years, with the greatest age incidence in the fifth and sixth decades. The condition has yet to be recognized at the Clinic in children 13 years of age or less. The duration of symptoms varied from a few days after the first renal colic to more than 30 years. There seems to be no correlation between the size or type of lesion and the duration of symptoms. Stones may form at any time during the course of the disease, whereas osteitis fibrosa requires a period of time to develop.

The values for serum calcium in the series varied from 10.1 to 18.4 mg/100 ml. of serum. Slight elevations were far commoner than extreme deviations from the normal (Figure). The value for calcium was less than 11 mg. in 13% of cases and less than 12 mg. in 54% of cases. At the other extreme, values of more than 14 mg. were found in less than 12% of cases. Although it is not the purpose of the present review to discuss the diagnosis of hyperparathyroidism, it could well be emphasized that its recognition still depends on the demonstration of hypercalcemia, and, as shown in the present series, the degree of hypercalcemia is often minimal indeed. From the standpoint of diagnosis, hypophospha-



The values for serum calcium and the incidence of osseous disease in proved cases of hyperparathyroidism, 1928-1954.

temia and hypercalciuria are of less importance. If renal insufficiency is present, the value for phosphate may be normal or elevated. Hypercalciuria may be absent under conditions of the Aub diet; however, increased calcium in the urine is not always diagnostic of hyperparathyroidism, as it occurs in many other conditions besides hyperparathyroidism. There is considerable interest at present in devising other tests for hyperparathyroidism. Among these, tests based on phosphate deprivation, on phosphate turnover, and on calcium loading might be mentioned. So far, none have proved clearly superior to determinations of serum calcium, although tests based on calcium loading seem promising.[9]

TABLE 2.—*Pathologic Findings in Proved Cases, 1928-1954*

Pathologic Findings	Cases	Per Cent	
Single adenoma	177	86	
Multiple adenomas	8	4	6
Polyendocrine adenomas	5	2	
Primary hyperplasia	15	7	
Carcinoma	2	1	
Total	207	100	

VARIATION IN PATHOLOGIC FINDINGS AND ITS INFLUENCE ON SURGICAL PROCEDURES

One of the chief difficulties in treatment results from variations in the pathologic lesions. The pathologic findings in the series are given in Table 2. As compared with previous reviews of smaller numbers of cases, the proportion of cases caused by single adenomas is somewhat increased and that resulting from multiple adenomas and primary hyperplasia is slightly decreased.[5] From the surgical standpoint, the fact that more than one parathyroid may be involved, in this series in 28 cases (13%), is of the utmost importance.

If the surgical procedure is terminated after the finding and removal of a single enlarged gland, the procedure will fail completely to cure the patient in all cases in which other glands are involved. Hyperparathyroidism is the same regardless of the pathologic lesion, and involvement of more than one gland cannot be predicted clinically, except in the case of polyendocrine adenomas, in which more than one gland is always involved. In other types of cases the only certain method of excluding multiple lesions is the identification of the four parathyroid glands. Variation in the number of parathyroid glands and the surgical approach to them will be discussed subsequently.

Single Adenomas.—Adenomas are recognizable by their more or less characteristic yellow-brown color. The external surface is smooth, but slight lobulation is usually evident. Typically, there are no adhesions between the capsule and surrounding structures. Cysts, hemorrhage, and calcification are found occasionally, particularly in the larger adenomas. In cases in which degenerative changes are marked, the capsule may be considerably thickened, the characteristic color lost, and adhesions may be present. Adenomas weighing more than perhaps 500 mg. are rather easily recognized and found, except when they have lost their characteristic color. These larger adenomas tend to be displaced by their growth into regions of loose areolar tissue where they are easily seen. Small tumors, however, tend to be caught within the sulci of the thyroid or between neighboring structures and so remain concealed. Fortunately, since degenerative changes are unusual in smaller adenomas, they tend to retain their characteristic color.

In practice, the greatest difficulty in the treatment of hyperparathyroidism has resulted from the small size of many adenomas. The variation in size of the 127 single adenomas in the series in which weight was recorded is shown in Table 3. The size is expressed in terms of weight because of the variability of shape. The smallest adenoma in the series weighed 25 mg., which is materially less than a normal parathyroid weighs. In almost one-fourth of the cases the adenoma weighed less than 250 mg. and in half of the cases less than 750 mg.

It would be of tremendous help to the surgeon if there were a relationship between the size of the adenoma and the severity of the

disease. This possibility was first studied by Castleman and Mallory [10] in 1935, and has since been studied in the Clinic series.* In general, some correlation does exist, and large adenomas are more likely than small adenomas to be associated with marked hypercalcemia and also with osteitis fibrosa. However, beyond this, the relationship is so crude that it is of little actual help. Values for calcium higher than 14 mg/100 ml. have been observed with adenomas weighing less than 300 mg., and values less than 11.5 mg. have been observed occasionally in cases in which the adenomas weighed 5 gm. or more.

Multiple Adenomas.—If the present series is typical, multiple adenomas involving more than one gland can be expected in some 6% of cases. Unless the patient has associated hyperinsulinism, involvement of more than two glands is most unusual. In two of the eight cases of multiple adenomas in the series three glands were enlarged, but the exact pathologic interpretation was somewhat confused because renal insufficiency was associated in both cases.[11]

TABLE 3.—*Weight of Single Adenomas in 127 Cases Resulting from Single Adenomas*

Weight, Gm.	Number	Per Cent
Less than 0.25	28	22
0.25-0.49	27	21
0.50-0.74	9	7
0.75-0.99	9	7
1.0-4.9	26	21
5.0-9.9	18	14
10 or more	10	8
Total	127	100

In five cases in the series the patients had a most unusual syndrome characterized by hyperparathyroidism and hyperinsulinism and in three of the five cases by a pituitary tumor also.[12] In this syndrome, referred to as "polyendocrine adenomas," more than one parathyroid is always involved, and as many as all glands may be involved. The adenomas tend to differ from the usual single adenoma in that the enlargement of the parathyroids apparently results from multiple nodules, a condition somewhat analogous to multiple adenomatous goiter.

* References 4 and 5.

The proper treatment of multiple adenomas is removal of all enlarged glands. If all four glands are involved, a viable portion of one gland may be preserved to prevent tetany. It is not known as yet whether the hyperparathyroidism will recur in such cases nor how much tissue should be saved.

Primary Hyperplasia.—This condition is found in somewhat less than 10% of cases of hyperparathyroidism. It is usually easily recognized grossly, since all the parathyroid glands are enlarged. The hyperplastic tissue is darker in color than the usual adenoma and more lobulated. Pseudopods are common. The different glands are enlarged asymmetrically, and all parathyroid tissue is always involved. In some cases the asymmetry is so marked that one or more of the glands may appear uninvolved.

The correct treatment is subtotal resection, preserving a fragment of vascularized tissue about three times the size of a normal parathyroid.[5] The finding of all hyperplastic tissue has not proved particularly difficult, provided that the true nature of the lesion was recognized at the time of the exploration. The preservation of a fragment of vascularized viable tissue has been less easy, and tetany has resulted in several of the 15 cases in the series.

Adenomas made up largely of transitional water-clear cells are rather common, and in at least five cases in the series the cytologic appearance of single adenomas was indistinguishable from that of primary hyperplasia. Such adenomas may be distinguished readily from primary hyperplasia by biopsy of a second gland.

Carcinoma.—Carcinomas are fortunately rare, accounting for probably not more than 0.5% to 1% of cases. Cope, Nardi, and Castleman,[13] in a report of four cases in 1953, stated that 33 acceptable cases had been reported. If stricter criteria of malignancy are adopted, this number would probably be reduced somewhat. Woolner, Keating, and one of us (Black) [11] were able to find reports (through 1951) of only six cases in which metastasis to lymph nodes or viscera had been reported. Pathologists particularly in-

terested in the problem are agreed that tumors capable of metastasizing may be indistinguishable histologically from presumably benign adenomas.† Benign adenomas are, however, never locally invasive.

Lesions exhibiting any fixation to surrounding structures, whether apparently from adhesions or from invasion, should be removed by radical resection of the gland without biopsy and without breaking into the lesion. Multiple local recurrences have developed in several cases in which the capsule was ruptured during removal.[13]

LOCATION OF PARATHYROID TISSUE

The second, and purely surgical, problem in the treatment of hyperparathyroidism has to do with the widespread region in which parathyroid tissue can occur. The superior gland arises from the fourth branchial pouch, near the lateral anlage of the thyroid. Migration during the developmental period is not so great as that of the inferior gland, which arises from the third pouch in close association with the thymus. The inferior gland migrates caudally lateral to the developing thyroid and superior gland and may accompany the thymus into the mediastinum. Theoretically, a gland may be found at any level along the course of its descent. In addition to aberrations in development, a parathyroid, particularly when enlarged, may be displaced caudally in the same manner as a low-lying adenoma of the thyroid becomes intrathoracic. If the gland, before adult or anatomic displacement, is situated well posteriorly, its displacement is toward the posterior superior mediastinum; if situated originally more anteriorly, it is displaced into the anterior superior mediastinum. Regardless of the type of displacement, the glands remain in the visceral compartment of the cervical fascia or within the extension of the same space in the mediastinum.

Since the beginnings of parathyroid surgery, variations in location of parathyroid glands have been emphasized. However, purely anatomic studies, such as that of Welsh[14] and particularly that of Gilmour,[15] have shown that the anatomy is reasonably constant. In the latter study more than 90% of superior glands were found above the lower third of the thyroid on its posterior or medial surface. Ninety-five per cent of inferior glands were found in the immediate vicinity of the lower poles of the thyroid, and in another 3% of cases, the glands were found between 3 and 6 cm. below the inferior poles. Widely displaced glands were, of course, found occasionally by both Welsh and Gilmour.

The location of the parathyroids in the present series of surgical dissections was entirely in keeping with that reported by Gilmour.[15] Indeed, if the location of the parathyroid glands was not reasonably constant, the finding of atrophic glands and of tiny adenomas would be extremely difficult, if not impossible. In considering the location of parathyroid tissue, the experience at the Clinic prior to 1942 should probably be omitted. The practice of identifying parathyroid glands individually had not been adopted, and in eight patients treated between 1928 and 1941 fruitless cervical dissections were carried out. During this period only 14 cases of hyperparathyroidism were proved. Between 1942 and 1954, the diagnosis was established during life, and cervical explorations were carried out in 191 cases. Adenomas were found within the thyroid in two cases. In all cases except 2 of the 191 cases, the hyperfunctioning tissue was found and removed under direct vision through the cervical incision. In the remaining two cases, adenomas were found on subsequent exploration of the anterior superior mediastinum. In at least four other cases the adenomas were located by tracing vascular pedicles downward into the mediastinum from a cervical incision. In a number of other cases the hyperfunctioning tissue could probably have been considered technically as mediastinal in location. However, in all cases it was found and removed without the necessity of formal mediastinotomy.

A third mediastinal exploration, not included in the present series, became necessary

† References 11 and 13.

this year (1955), when possibly another 25 patients have been seen. In two of the three patients in whom mediastinal exploration was necessary, cervical exploration had been carried out previously elsewhere. Thus, of more than 200 patients treated primarily at the Clinic, in only one was formal mediastinotomy necessary to find the adenoma. In view of the rarity of tumors which could not be found and removed through the cervical approach, the policy of advising reexploration of the neck has been adopted in patients referred to the Clinic after negative cervical explorations elsewhere. An exception might be made in cases in which three glands have been proved by biopsy and the fourth proved missing after a reasonably extended search. No such case has been seen as yet.

The rarity of cases in which mediastinotomy was required in the series is somewhat difficult to reconcile with an incidence of 10% to 15% of such cases reported by others. The size of the adenoma may have some bearing on the point. Small adenomas, which characterize the present series, are unquestionably far less likely to be displaced into the mediastinum than larger tumors, such as those in the collected series of Norris.[16] If this is the explanation, then anatomic or adult displacement is far commoner than is displacement during the developmental period.

TECHNICAL ASPECTS

The most successful approach to the problems posed by the variations in the pathologic lesions and the occasional mediastinal adenoma is the methodical identification of each of the four parathyroid glands. There is, of course, some variation in the number of parathyroids. Fewer than four glands were found in slightly more than 6% of Gilmour's[15] dissections in 428 necropsy cases and in more than four glands in another 6% of cases. When only two or three glands were found, the total weight of parathyroid tissue suggested that glands had been missed in the dissection. In other cases the two glands on one side were fused, a condition first described by Welsh.[14] When more than four glands were present, the supernumerary glands were small and in the immediate vicinity of a normal gland. If an adenoma developed in such a supernumerary gland, it would not be apparent whether the adenoma arose in the normal or supernumerary gland, nor would the adenoma cause any difficulty surgically. For practical purposes the possibility of an adenoma developing in a widely aberrant supernumerary gland seems remote. There were no such cases in the present series.

The presence of a pathologist competent to recognize parathyroid tissue and the pathologic lesions which may be encountered from the examination of fresh-frozen sections is most essential. The recognition grossly of atrophic parathyroids, regardless of the surgeon's experience, is often uncertain. As previously mentioned, wasserhelle-cell adenomas must be differentiated from primary or wasserhelle hyperplasia. Finally, it is most reassuring to have histologic proof, before the incision is closed, that the lesion removed is, in fact, the lesion responsible for the disease.

Biopsies of uninvolved glands are indicated infrequently. In the usual case in which an adenoma is found, gross identification of uninvolved glands is adequate. If the adenoma is of the wasserhelle-cell type, a biopsy from an uninvolved gland, as previously mentioned, will aid the pathologist materially in making the correct diagnosis. Biopsies of uninvolved glands become mandatory only in those cases in which a lesion has not been found, to indicate with certainty into which regions the dissection should be extended in the search for the missing gland. In order to avoid destruction of the gland, extremely minute specimens should be taken and no effort should be made to control the slight bleeding which results. Specimens should never be taken from grossly enlarged glands; instead, such glands should be removed completely without rupturing the capsule. As mentioned previously, in the extremely unusual case of polyendocrine adenomas with involvement of four glands and in cases of wasserhelle hyperplasia subtotal resection should be carried out to avert chronic tetany.

Exploration of the mediastinum should be undertaken only in cases in which there is reasonable certainty that the adenoma cannot be removed through the cervical incision. Before this can be known, the parathyroid glands in the cervical region must be found and verified by biopsy. Ideally, three glands should be found and the fourth proved missing by an extended search of the appropriate region. While this ideal may not be achieved, the number of glands found is a measure of the completeness of the cervical dissection. We are in complete agreement with Cope that the mediastinum should be explored at a subsequent time and not in conjunction with the cervical exploration. A cervical dissection sufficiently complete to exclude an adenoma requires several hours, and another prolonged surgical procedure seems inadvisable at that time from the standpoint of both surgeon and patient. The presence of hyperparathyroidism should probably be verified again by further chemical tests and any tissue removed at time of exploration restudied before mediastinal dissection is undertaken. One patient in the series was cured after what appeared at the time to be a negative cervical exploration at thyroidectomy. However, an adenoma of the parathyroid was removed with the thyroid tissue but was not recognized until later when the tissue was restudied.

SUMMARY AND CONCLUSIONS

Two hundred seven cases of hyperparathyroidism in which the diagnosis was proved by pathologic examination of tissue removed at operation or at necropsy have been seen at the Mayo Clinic through 1954. Complications involving the urinary tract were present in 82% of cases, and osseous complications were found in 29%. In 6% of cases the diagnosis was made in the absence of such complications.

In 86% of cases the disease resulted from a single adenoma, in 6% from multiple adenomas, in 7% from primary hyperplasia, and in 1% from carcinoma.

The adenomas in the series varied in weight from 25 mg. to 101 gm. Fifty per cent of single adenomas weighed less than 750 mg. The most frequently encountered problem in treatment was the finding of small adenomas. Another less serious difficulty had to do with the fact that more than one parathyroid gland was involved in 13% of cases.

The location of parathyroid glands is far more constant than has been generally assumed. The great majority of adenomas are in the immediate vicinity of the thyroid and may be removed under direct vision through a cervical incision. Formal exploration through a sternotomy was necessary in less than 0.5% of the patients on whom the primary operation for hyperparathyroidism was performed at the Mayo Clinic since 1941.

The surgical procedure recommended is the methodical identification of the four parathyroid glands. The mediastinum should not be explored until the parathyroids found on cervical incision have been identified by biopsy and any unidentified glands have been searched for extensively and not found.

DISCUSSION

Dr. Gordon S. Fahrni, Vancouver, B. C., Canada: It is a great privilege to have the opportunity to come up to this rostrum and open the discussion of Dr. Black's paper and to thank him for having given us his review of such a tremendous number of cases.

I have had the opportunity of having nine proved cases, all of which were adenomas and all of which had skeletal decalcification and two of which had renal lithiasis also.

I should like to ask Dr. Black if he differentiates at all between the parathyroid adenomas and the diffuse hyperplastic type, which to me seems to be a different disease.

In referring to the group of nine parathyroid adenomas among nine female patients from 11 to 63 years of age, I have picked out just a few features to demonstrate some points that I have learned and that I believe are important.

The first point is that there are two types of decalcification seen in hyperparathyroidism: (*a*) diffuse decalcification in one or more parts of the skeleton and (*b*) localized areas in the form of giant-cell tumors spotted about the decalcified skeleton. The microscopic picture of these expanded bone lesions are indistinguishable from the classical giant-cell tumor. In nine cases I have had three with multiple giant-cell tumors, usually situated in the ends of long bones, in addition to the presence of diffuse decalcification.

I should like to stress the importance of correcting any gross deformity just after the parathyroid tumors are removed and before recalcification takes place, i. e., while the bones are still pliable. Two examples are severe coxa vara, where the upper end of femurs are involved, and compression fractures of vertebral bodies not uncommon in decalcification of these bones.

In our nine cases there were three each with 2 parathyroid tumors, 12 tumors in all. There were three parathyroid adenomas in the anterior mediastinum and one intrathyroid adenoma. In one of our twin-tumor cases, one tumor was in the anterior mediastinum and the other was intrathyroid. The embryological explanation for these aberrant sites is readily apparent when we recall that the inferior parathyroid arises with the thymus gland from the third branchial pouch and the superior arises with the lateral thyroid from the fourth branchial pouch.

DR. B. MARDEN BLACK, Rochester, Minn.: In answer to the question regarding the differences in the clinical manifestations of hyperparathyroidism, as far as we know there is no difference clinically between the hyperparathyroidism that results from primary hyperplasia and that caused by an adenoma. Parathyroid adenomas do occur within the thyroid gland. The incidence in the Clinic series is approximately 1%. Larger adenomas unquestionably tend to be displaced into the mediastinum more frequently than the tiny adenomas which characterize the Clinic series. At least this is the only explanation I have for the marked discrepancy between the location of the adenomas in the present series and those reported in the literature.

REFERENCES

1. Churchill, E. D., and Cope, O.: Parathyroid Tumors Associated with Hyperparathyroidism: Eleven Cases Treated by Operation, Surg. Gynec. & Obst. **58**:255 (Feb. 15) 1934.

2. Cope, O.: Surgery of Hyperparathyroidism: The Occurrence of Parathyroids in the Anterior Mediastinum and the Division of the Operation into 2 Stages, Ann. Surg. **114**:706 (Oct.) 1941.

3. Rienhoff, W. F., Jr.: The Surgical Treatment of Hyperparathyroidism, with a Report of 27 Cases, Ann. Surg. **131**:917 (June) 1950.

4. Black, B. M.: Surgical Aspects of Hyperparathyroidism: Review of 63 Cases, Surg. Gynec. & Obst. **87**:172 (Aug.) 1948.

5. Black, B. M.: Hyperparathyroidism, Springfield, Ill., Charles C Thomas, Publisher, 1953.

6. Barr, D. P.; Bulger, H. A., and Dixon, H. H.: Hyperparathyroidism, J. A. M. A. **92**:951 (March 23) 1929.

7. Keating, F. R., Jr., and Cook, E. N.: The Recognition of Primary Hyperparathyroidism: An Analysis of 24 Cases, J. A. M. A. **129**:994 (Dec. 8) 1945.

8. Albright, F.; Aub, J. C., and Bauer, W.: Hyperparathyroidism: A Common and Polymorphic Condition as Illustrated by Seventeen Proved Cases from One Clinic, J. A. M. A. **102**:1276 (April 21) 1934.

9. Howard, J. E.; Hopkins, T. R., and Connor, T. B.: On Certain Physiologic Responses to Intravenous Injection of Calcium Salts into Normal, Hyperparathyroid and Hypoparathyroid Persons, J. Clin. Endocrinol. **13**:1 (Jan.) 1953.

10. Castleman, B., and Mallory, T. B.: The Pathology of the Parathyroid Gland in Hyperparathyroidism: A Study of 25 Cases, Am. J. Path. **11**:1 (Jan.) 1935.

11. Woolner, L. B.; Keating, F. R., Jr., and Black, B. M.: Tumors and Hyperplasia of the Parathyroid Glands: A Review of the Pathological Findings in 140 Cases of Primary Hyperparathyroidism, Cancer **5**:1069 (Nov.) 1952.

12. Underdahl, L. O.; Woolner, L. B., and Black, B. M.: Multiple Endocrine Adenomas: Report of 8 Cases in Which the Parathyroids, Pituitary and Pancreatic Islets Were Involved, J. Clin. Endocrinol. **13**:20 (Jan.) 1953.

13. Cope, O.; Nardi, G. L., and Castleman, B.: Carcinoma of the Parathyroid Glands: Four Cases Among 148 Patients with Hyperparathyroidism, Ann. Surg. **138**:661 (Oct.) 1953.

14. Welsh, D. A.: Concerning the Parathyroid Glands: A Critical, Anatomical, and Experimental Study, J. Anat. & Physiol. **32**:292 (Jan.): 380 (April) 1898.

15. Gilmour, J. R.: The Gross Anatomy of the Parathyroid Glands, J. Path. & Bact. **46**:133 (Jan.) 1938.

16. Norris, E. H.: Collective Review: The Parathyroid Adenoma: A Study of 322 Cases, Internat. Abstr. Surg. **84**:1, 1947; in Surg. Gynec. & Obst., Jan., 1947.

Congenital Vascular Lesions Imitating the Patent Ductus

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In the majority of patients with patent ductus arteriosus, with its associated continuous murmur and other classic findings, the diagnosis is usually not difficult, and the surgical treatment in these cases has become quite standardized. In doubtful cases catheterization and aortography are most helpful. There are, however, a few cardiovascular lesions which may be confused with a patent ductus arteriosus. In a series of 100-odd cases, operated upon at the Cook County Children's Hospital and the Presbyterian Hospital, certain vascular lesions were found which simulated a patent ductus. The purpose of this paper is to evaluate these conditions and to present the related problems.

TABLE 1.—*Cardiovascular Lesions Imitating the Patent Ductus Arteriosus*

1. Arteriovenous fistula
   a. Pulmonary
   b. Subclavian
   c. Anomalous coronary circulation
   d. Internal mammary
   e. Intercostal
2. Aortic septal defect
3. Aneurysm of sinus of Valsalva with rupture into right ventricle or atrium
4. Aneurysm of aorta with rupture into pulmonary artery
5. Interventricular septal defect

Read at the 63d Annual Meeting of the Western Surgical Association, Seattle, Dec. 2, 1955.

From the Department of Surgery, The Presbyterian Hospital, and The University of Illinois College of Medicine (Rush), and the Cardiac Division of the Children's Cook County Hospital (Hektoen Research Laboratory).

REPORT OF CASES

CASE 1.—A 4-month-old infant was admitted on Dec. 30, 1953, to the Cook County Children's Hospital because of difficulty in breathing. She had experienced a recent upper respiratory infection. There was no history of cyanosis.

Physical examination revealed a well-developed infant who was not cyanotic but was very dyspneic. The lungs were clear. The heart was enlarged to the left midclavicular line, fourth interspace. There was a blowing systolic murmur heard at the base of the heart without evidence of a diastolic murmur; however, phonocardiography, with the patient under sedation, demonstrated a diastolic component. P_2 was greater than A_2. Blood pressure was 90/50 in the right arm. Examination of the abdomen revealed that the liver was 2 fingerbreaths below the costal margin. The spleen was just palpable.

Examination of the blood revealed hemoglobin of 68%, a red blood cell count of 4,200,000, and a white blood cell count of 22,000. The differential count was normal. The roentgenologic examination of the chest revealed moderately congested lung fields and an enlargement of the heart due to left ventricular

Fig. 1.—Preoperative roentgenogram of the chest, showing enlarged heart and increased pulmonary vascular markings.

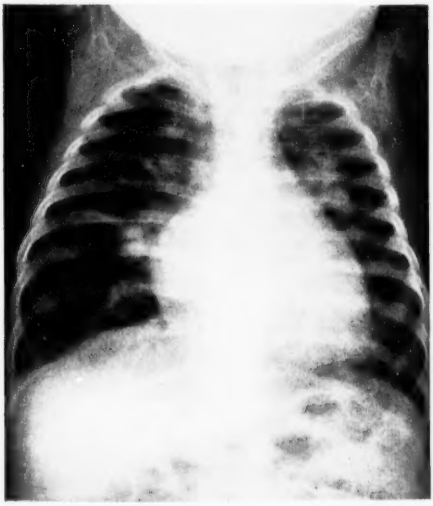


TABLE 2.—*Heart Catheterization Data in Aortic-Pulmonary Fistula*

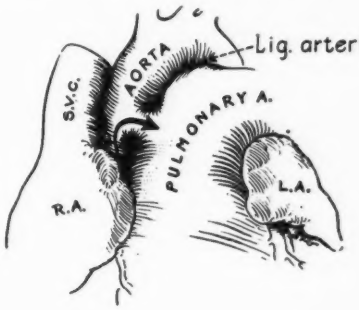
	Oxygen Content, Vol. %	Intravascular Pressures, Mm. Hg	Oxygen Saturation
Inferior vena cava	5.61	.....	....
Right atrium	6.08	2	....
Right ventricle (inflow)	6.65	.....	....
Right ventricle (outflow)	6.88	.....	....
Pulmonary artery (main)	10.54	82/49	73%
Pulmonary artery (left)	9.75	.....	....
Innominate artery	....	88/52	....
Calculated blood flows	Systemic 0.59 L/min. Pulmonary 1.08 L/min.	} Left to right shunt = 0.49 L/min.	

hypertrophy (Fig.). Catheterization studies (Table 2) revealed moderate hypertension in the right ventricle and the pulmonary artery with a marked elevation of oxygen content in the pulmonary artery. The angiocardiogram demonstrated a large pulmonary artery. The aorta and the right common carotid artery were entered by way of the pulmonary artery, providing evidence of a communication between the lesser and the greater circulation (Fig. 2). The course of the catheter suggested primarily an aortic septal defect, but a patent ductus arteriosus could not be definitely excluded.

On March 12, 1954, a left thoracotomy was performed. No patent ductus arteriosus was found. Further exploration revealed a prominent thrill over a markedly dilated pulmonary artery. Additional dissection disclosed an aortic-pulmonary fistula (Fig. 3). The tract was dissected out very carefully and ligated with two heavy nonabsorbable surgical (silk) suture ligatures. The thrill in the pulmonary artery disappeared immediately after closure of the defect. After a stormy convalescence, the patient improved sufficiently to be discharged from the hospital.

On Oct. 24, 1955, the patient was reexamined in the outpatient department. She was gaining strength and weight and showed evidence of growth. Examination of the heart revealed at the base a residual systolic murmur, possibly due to the still dilated pulmonary artery.

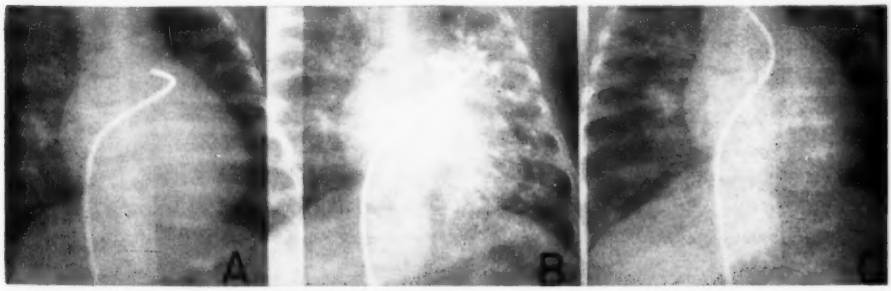
CASE 2.—A 3-year-old girl was admitted to the Cook County Children's Hospital on May 11, 1953, with a history of numerous episodes of transient and



Congenital Aortic-pulmonary Fistula

Fig. 3.—Drawing of communication between the aorta and markedly dilated pulmonary artery. Two suture ligatures were used to close the fistula in continuity.

Fig. 2.—Retrograde catheter (via right saphenous vein) is observed in the pulmonary artery (*A*), visualization of left vascular lung field with sodium acetrizoate (Urokon) (*B*), and catheter passing through the pulmonary artery into the aorta and inominate artery (*C*).



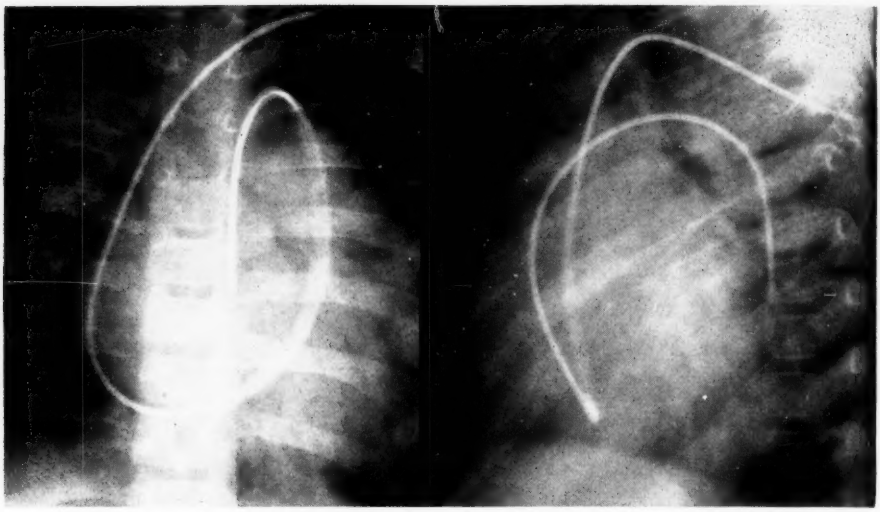


Fig. 4.—Catheter passed into the right side of the heart and pulmonary artery is directed into the descending aorta by way of a large patent ductus arteriosus.

intermittent cyanosis since the age of 13 months. There was a noticeable progressive dyspnea for the previous six months.

Physical examination showed an acutely ill child who was markedly dyspneic and was coughing constantly. There was no evidence of cyanosis, clubbing, or edema. The child was febrile. Examination of the lungs revealed moist crepitant rales bilaterally. The heart was enlarged to the left midaxillary line in the sixth interspace. There was a thrill palpable over the upper precordium. There was a continuous machinery-like systolic and diastolic murmur over the first and second interspaces on the left, as well as a similar murmur over the first and second right interspaces. These murmurs were transmitted to the back on both sides of the chest, loudest over the right posterior chest. The blood pressure in the right arm equalled 104/60.

The laboratory data were as follows: No anemia was present. The urine was negative. Fluoroscopic examination of the chest showed marked enlargement of the heart in all views, with a bulging pulmonary artery. The lung fields were hazy and mottled. The pulmonary vessels appeared very large, but no hilar dance was noted. The electrocardiogram showed left and right ventricular hypertrophy, as well as left auricular hypertrophy. The angiocardiogram demonstrated marked filling of the lung fields and reopacification of the main pulmonary artery. The catheterization data showed that there was an increase in the oxygen content of the right ventricular outflow tract and of the main pulmonary artery. A catheter was passed into the descending aorta via a patent ductus on the left side (Fig. 4). The clinical impression, therefore, was patent ductus arteriosus on the left side. However, in view of the loud machinery-like murmur over the right side of the chest, the possibility of additional pathology, such as a right patent ductus arteriosus or a collateral arterial vessel or a communication be-

TABLE 3.—*Heart Catheterization Data in Postoperative-Patent Ductus Arteriosus*

	Oxygen Content, Vol. %	Intravascular Pressures, Mm. Hg	Oxygen Saturation
Superior vena cava	7.11	.....	....
Right atrium	8.65	4	....
Right ventricle (inflow)	9.05	.....	....
Right ventricle (outflow)	10.62	73/0	....
I. V. septal defect (ventricle)	12.37	86/1	....
Aorta	12.43	86/63	96.1%
Calculated blood flows	Systemic 1.77 L/min.	} Left to right shunt = 2.49 L/min.	
	Pulmonary 4.26 L/min.		

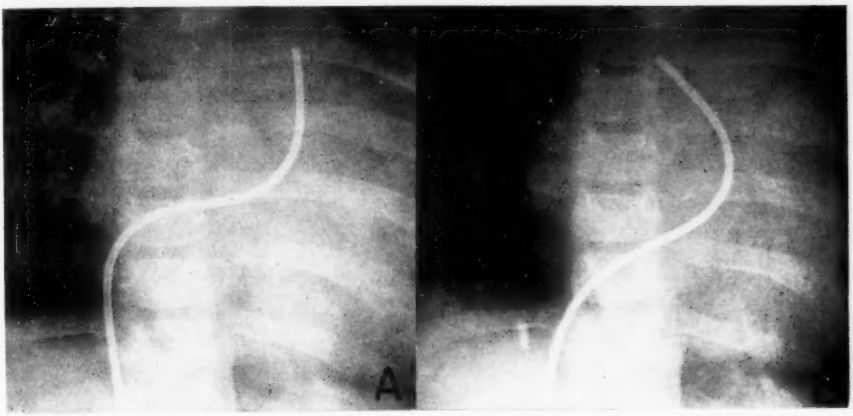


Fig. 5.—Postoperative catheterization through the right saphenofemoral junction showed a marked degree of hypertension in the right ventricle and an increased oxygen content. The catheter is visualized in the pulmonary artery (*A*) and in the aorta (*B*) by way of an interventricular septal defect.

tween the aorta and the right ventricle, such as a ruptured sinus of Valsalva, could not be excluded.

On July 28, 1953, at operation a large patent ductus was found on the left side. The ductus was dissected out and closed with multiple suture ligatures. During the procedure, cardiac arrest occurred; cardiac massage restored the heart to normal activity. The postoperative course was uneventful. At the time of discharge the continuous machinery-like murmur over the right chest was still in evidence.

On Oct. 2, 1953, the patient was readmitted for treatment of acute pneumonitis. Catheterization studies were repeated (Table 3). There was still an elevated oxygen content and high pressure in the right ventricle. The catheter was passed through an interventricular septal defect into the aorta (Fig. 5). However, because of the previously described continuous systolic and diastolic murmurs over the right second interspace and the relatively high pulse pressure, it was felt that an exploratory thoracotomy on the right side was indicated.

On Dec. 8, 1953, the right chest was opened and no extracardiac pathology was found to explain the murmur. The pericardial cavity was not explored. A large pulmonary artery with increased pressure was observed. No further surgery was done at this time, and no satisfactory explanation of the continuous murmur was apparent. Retrograde aortography was advised, but the parents objected to further procedures at that time. We hope to study this patient further.

CASE 3.—A 19-year-old woman was admitted to the Cook County Hospital on May 16, 1953, for treatment of an upper respiratory infection. An incidental finding on examination of the chest was a continuous machinery-like murmur, heard best in the pulmonic area. No other murmurs were heard. The heart was normal in size, with a regular rate and rhythm. The blood pressure was 110/80; the pulse rate was 76 per minute. The remainder of the physical examination was not unusual.

Laboratory examination of the blood revealed hemoglobin of 80%, red blood count of 4,100,000 and white blood cell count of 6150. The urinalysis was negative. Blood cultures were negative. The x-ray

Fig. 6.—Preoperative chest film demonstrates a normal sized heart and relatively normal appearing vascular markings in the lung fields.

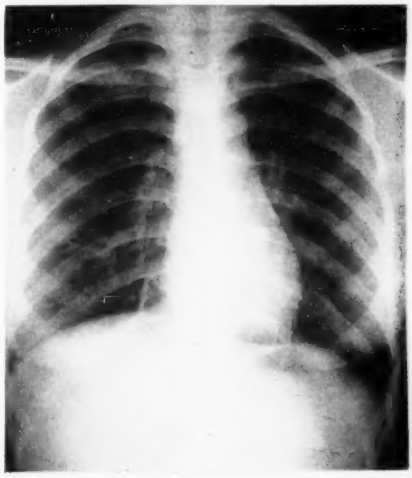


TABLE 4.—*Heart Catheterization Data in Anomalous Coronary Artery*

	Oxygen Content, Vol. %	Intravascular Pressures, Mm. Hg	Oxygen Saturation
Superior vena cava	11.73	.....	....
Right atrium	12.44	2	....
Right ventricle (inflow)	12.47	.....	....
Right ventricle (outflow)	14.07	22/2	....
Pulmonary artery (main)	13.29	19/9	....
Right femoral artery	16.67	114/68	98.3%
Calculated blood flows	Systemic 5.10 L/min. Pulmonary 6.22 L/min.	Left to right shunt = 1.12 L/min.	

examination of the chest revealed a normal-sized heart (Fig. 6). No increased pulsations were observed in the pulmonary artery. The electrocardiogram was normal. The catheterization studies (Table 4) indicated an oxygen content of 14.07 vol. % in the outflow tract of the right ventricle. This was 1.5 vol. % higher than the more proximal samples and 0.7 vol. % less than the oxygen content of 13.29 vol. % in the main pulmonary artery. These findings were consistent with the left-to-right shunt at the ventricular level—or they were suggestive of a patent ductus arteriosus with pulmonary regurgitation.

On June 4, 1953, the patient was operated upon for correction of a patent ductus arteriosus. At operation only a ligamentum arteriosum was found. In the search for the cause of the continuous murmur, the pericardial sac was opened longitudinally. A most unusual condition was found (Fig. 7). A very large tortuous left coronary artery was observed. The vessel circumvented the anterolateral aspect of the left atrium and anteriorly traversed the atrioventricular groove, giving off small branches to supply the myocardium of the left ventricle. There was no vessel identified as the circumflex branch of the left coronary artery. The more distal course of the anomalous artery imitated an S-curve as it crossed the interventricular septum to terminate in the infundibular area of the right ventricle just proximal to the pulmonary valve. Here a definitely palpable thrill was present. Temporary occlusion of the vessel just proximal to its entrance into the right ventricle resulted in an immediate disappearance of the thrill. Therefore, closure of the "shunt" was accomplished by removing a small section of the artery distal to the last descending branch. Hemostasis was obtained with suture ligatures. As soon as this procedure was completed,

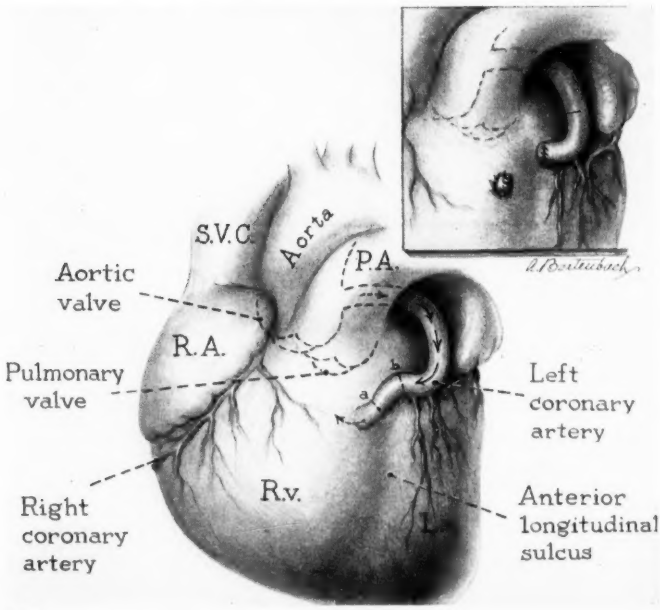


Fig. 7.—Drawing of anomalous left coronary artery showing the course of the vessel and the method of treatment of the "arteriovenous shunt."

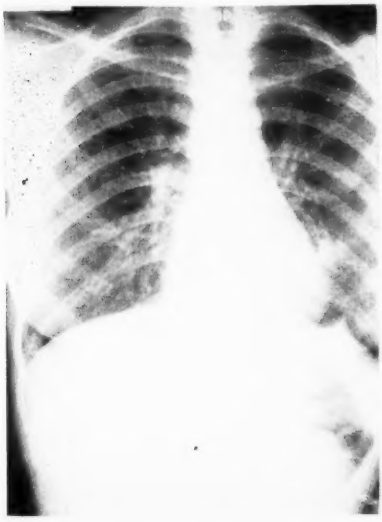
the thrill disappeared and the apparent increased tension in the right ventricle decreased. No cardiac irregularity was noted.

The postoperative course was complicated by persistent tachycardia, a pericardial friction rub, pericardial effusion, and a mild pulmonary infection. The continuous murmur, however, was gone. The patient was discharged in good condition 35 days after the thoracotomy.

Approximately 15 months later, on Oct. 27, 1954, the patient was reexamined. There were no symptoms referable to her cardiac status or to the previous surgery. She was well and had gained about 10 lb. of weight. Examination of her chest revealed a normal cardiac rhythm without evidence of any murmurs. The blood pressure was 110/70; pulse rate was 76 and regular. A postoperative roentgenogram (Fig. 8) of the chest showed very little change in heart size as compared with the preoperative one. The electrocardiogram was unchanged from that of the admission record.

CASE 4.—A 43-year-old man was admitted to the Presbyterian Hospital on Jan. 30, 1955. He was actually referred to the hospital for surgical treatment of a presumed mitral stenosis. This patient had been an active laborer for many years until he had experienced symptoms referable to his heart during the previous two years. His complaints consisted of easy fatigue, a heavy pressure in the chest, a chronic cough, shortness of breath, and palpitation of the heart. There was no history of sudden acute cardiac pain. He stated that he had a known heart murmur of at least 10 years' duration. Twenty years ago he had been examined by an insurance doctor and no murmur was found at that time; at least he was given an insurance policy.

Fig. 8.—Postoperative roentgenogram (Oct. 27, 1954) compares favorably with preoperative film.



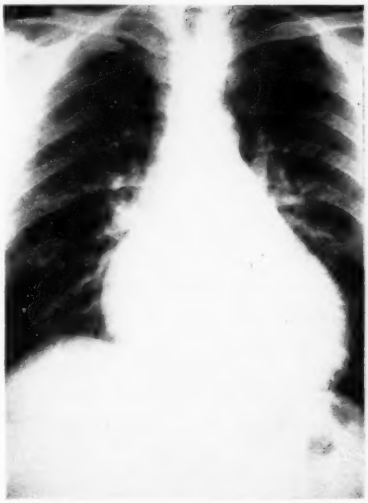


Fig. 9.—Roentgenogram of the chest showing marked enlargement of the heart in all directions and increased pulmonary vascular markings.

Physical examination revealed a middle-aged man who was somewhat apprehensive about his condition. Mild dyspnea was present. A hacking cough was noted. The neck veins were not dilated. There was no cyanosis. Blood pressure was 150/60. Pulse rate was 80 and regular. Respirations were 16 per minute. Examination of lungs and abdomen was negative. The examination of the heart was most interesting. The heart was enlarged in the anterior axillary line in the left sixth interspace. There was a low-pitched Grade 4 systolic murmur, heard loudest in the fourth interspace on the left, and a similar murmur heard in the apical region through to the back. The systolic murmur was continuous with a high-pitched diastolic murmur heard over the left side of the sternum, loudest in the fourth and fifth interspaces.

Laboratory examination revealed hemoglobin of 13 grams, red blood cell count 4,200,000, and white blood cell count 5100. Kahn test was negative. The urine was negative. An anteroposterior view of the chest showed that the heart was markedly increased in size. There was an increase in vascular markings in both lungs (Fig. 9). Catheterization studies (Table 5) indicated a moderate elevation of pressure in the outflow tract of the right ventricle and of the pulmonary artery. Of interest were the oxygen content levels; that of the superior vena cava was 12.42 vol. %, of the right auricle 15.80 vol. %, of the right ventricle 15.83 vol. %, and of the pul-

TABLE 5.—*Heart Catheterization Data in Rupture of Sinus of Valsalva (Right Atrium)*

	Oxygen Content, Vol. %	Intravascular Pressures, Mm. Hg
Superior vena cava	12.42	.....
Right atrium (inflow)	14.99	.....
Right atrium (outflow)	15.80	.....
Right ventricle (outflow)	15.80	42/10
Pulmonary artery	15.97	41/7
Peripheral artery	17.86	122/52
% saturation	96%	

Pulmonary blood flow was 2.8 times as great as systemic flow

monary artery 15.97 vol. %. The peripheral arterial oxygen content was 17.86 vol %. The pulmonary blood flow was 2.8 times as great as the systemic flow.

As a result of these data, an interauricular septal defect, anomalous pulmonary veins, or some other rare intracardiac anomaly was considered. The murmurs were not characteristic of an atrial septal defect; the continuous quality of the murmurs, along with the wide pulse pressure and the evidence of right ventricular hypertrophy on the electrocardiogram suggested a patent ductus, or some communication between the aorta and the right atrium or ventricle.

On Feb. 10, 1955, a right thoracotomy was carried out through the bed of the resected fifth rib. A huge right atrium was found which contained bright-red blood, suggesting a left to right shunt. Exploration of the right side of the heart through the auricular appendage suggested a high-lying interventricular septal defect with possible involvement of the atrial wall. A definite jet of blood was palpated approximately at the level of a dilated tricuspid valve ring. Additional surgery was deemed inadvisable at this time. After a not too unusual postoperative course, the patient died suddenly and unexpectedly on the 10th postoperative day.

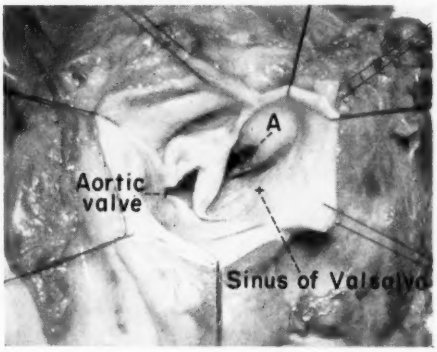
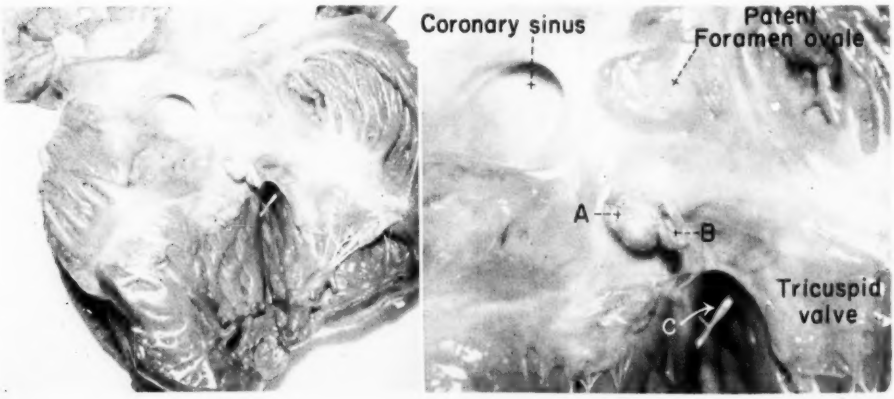


Fig. 11.—Cross section of aorta shows the aortic valve and the opening of the aneurysm in the floor of the right sinus of Valsalva at *A*.

The autopsy examination of the heart revealed some interesting findings (Fig. 10). Within the septum of an enlarged heart (weight 570 gm.) there was a communication between the aorta and the right auricle. The origin of this tract was in the floor of the right sinus of Valsalva (Fig. 11), while the exit involved a small projection of endocardium with orifices or perforations (Fig. 10)

Fig. 10.—Left, photograph of the right side of the heart illustrating the markedly dilated atrium and ventricle. The small aneurysmal projection is located in the atrium, just above the level of the tricuspid ring. Right, sites of apparent rupture of aneurysm of sinus of Valsalva at *A* and *B*. Aneurysm lies just above base of the tricuspid valve. Outflow tract of right ventricle seen behind chorda tendinea at *C*.



through which the arterial blood was propelled into the right atrium. This vascular malformation was considered to be of congenital origin rather than due to trauma. It is possible that under hypothermia with open cardiotomy successful surgical closure might have resulted.

COMMENT

A variety of vascular conditions may imitate the characteristic continuous machinery-like murmur of a patent ductus arteriosus. Venous hums have been described which imitate the murmur of a patent ductus, but these are usually obliterated readily by compression of the jugular vein. The murmur is transmitted from the neck down to the chest, and the diastolic phase of the murmur is usually accentuated.

An aortic septal defect or aortic-pulmonary fistula usually produces a continuous murmur and may simulate a patent ductus arteriosus. Only in recent years has this lesion been attacked surgically. At the present time, very few discriptions of this lesion have appeared in the surgical literature. In 1951, Gasul, Fell, and Casas [1] described an aortic septal defect in a 3½-month-old infant. This malformation was diagnosed by retrograde aortography. In 1949, Dadds and Hoyle [2] reviewed extensively the subject of congenital aortic septal defect and stated that in none of the recorded cases was the diagnosis made in life. The case of a 14-year-old lad with the clinical findings of an aortic septal defect was described in detail. They believed that during life they were dealing with a complicated defect including a patent ductus arteriosus and a possible atrial septal defect; however, at autopsy, there was found a large aneurysmal sac compressing the main pulmonary artery and the contiguous aorta. The aneurysmal sac when opened showed a wide communication (6.0×5.0 cm.) between the hugely dilated pulmonary artery and the base of the aorta just above the valves. A huge septal defect of this type would not readily lend itself to surgical closure without extreme risk.

In 1950, Gibson, Potts, and Langewisch [3] reported four cases of aortic septal defects in children who had preoperative findings compatible with that of a patent ductus, but in all instances only a ligamentum arteriosum was found. In 1953 Holman, Gerbode, and Purdy,[4] in a review of their experiences with the patent ductus arteriosus described a few cases which were diagnosed preoperatively but in which no such lesion was found at operation. Aortic septal defects or high interventricular septal defects were suspected. At autopsy in the fifth patient a high interventricular septal defect with an aneurysm of the sinus of Valsalva, communicating with the right ventricle, was found. This case was further complicated by bacterial endocarditis.

In 1950, Spencer and Dworken [5] wrote "in view of the present success of cardiac surgery, it is possible that aortic septal defects may be relieved by operation." In 1953, Scott and Sabiston,[6] in the *Journal of Thoracic Surgery*, discussed the problems involved in the surgical treatment of aortic-pulmonary fistula or aortic septal defect and described two cases of their own. One patient succumbed without surgery, and at autopsy an aortic-pulmonary fistula was found. In the second case the fistulous tract was treated successfully with division between clamps and closure accomplished with two rows of 00000 nonabsorbable surgical (silk) sutures. Certain interesting points regarding the technique are described. In the discussion of the above-mentioned paper, Robertson * stated that in operating upon 45 patients suspected of having a patent ductus there were four instances of aortic pulmonary communications for which no surgery was undertaken. Gross and Varco † are reported to have accomplished successful closures of aortic-pulmonary fistula in individual cases by ligation or suture ligation.

Our second case continues to bring about speculation as to the cause of the continuous murmur on the right side. It is apparent that an interventricular septal defect is present, but a communication between the aorta and

* Robertson, R., in discussion of Scott and Sabiston.[6]

† Gross and Varco, cited by Scott and Sabiston.[6]

the right ventricle has not been ruled out. The second thoracotomy was performed to rule out a right patent ductus on the basis of a report by Kelsey, Gilmore, and Edwards,[7] who, in 1953, described the case of a 10-month-old boy who at autopsy was found to have a bilateral ductus arteriosus, associated with an isolated dextrocardia, ventricular septal defect, and pulmonary atresia. The left ductus was obliterated, while the right ductus arteriosus was patent. This malformation of a bilateral ductus arteriosus is caused by the abnormal retention of the elements of the sixth (pulmonary) aortic arch.

In 1952, Dammann and Sell [8] reported on 24 patients, each one suspected of having a patent ductus arteriosus but without the characteristic continuous machinery murmur. Seven of these cases were diagnosed at surgery, in the absence of a patent ductus, as having high interventricular septal defects. One infant 4½ months of age succumbed after surgical exploration of a presumed patent ductus and, at autopsy, there was observed a closed ductus associated with a high-lying ventricular septal defect and an overriding aorta. These authors emphasize that catheterization studies and retrograde aortography are of great assistance in differentiating the patent ductus from other conditions which tend to imitate it. Gasul, Fell, Dillon, and Marienfeld [9] reported on 14 infants less than one year of age who entered the hospital in heart failure with an "atypical, malignant" type of patent ductus areriosus. Three of these patients, who were not operated upon, died. The remaining 11 were all successfully operated upon. A continuous murmur was heard in 50% of these patients. All of these infants presented a high pulse pressure. The authors stressed the great importance of the presence of high pulse pressure in the absence of a continuous murmur as a very suggestive sign of patent ductus arteriosus in these infants. In all of these cases cardiac catheterization and/or retrograde aortography are definitely indicated.

The presence of an anomalous left coronary artery supplying blood to the infundibular area of the right ventricle provides an unusual explanation for a left-to-right shunt. In a previous paper [10] we discussed certain congenital anomalies or acquired defects which are confused with the patent ductus. White [11] states that anomalies of the coronary arteries are reported rather infrequently. The major anomaly concerns the left coronary artery which has originated from the pulmonary area, with death occurring in most instances during infancy. Moreover, reports of coronary vessels communicating directly with ventricular cavities are infrequent. In 1951, Williams, Kent, and Edwards [12] reported the autopsy findings in a 4-day-old infant in whom an anomalous coronary vessel communicated with the chambers of the right ventricles by means of sinusoids and emerged from the wall of the right ventricle to pass inferiorly and anastomose with the posterior descending coronary artery. From this point its course was downward over the apex, where it anastomosed with a branch of the left circumflex coronary artery. In 1952, Alexander and Green [13] described in a newborn infant an anomalous vessel which took origin from the left coronary artery and communicated with the lumen of the right ventricle. Pulmonary valvular atresia and a small interventricular septal defect were also present. In these two cases it was suggested that the anomalous coronary blood vessel developed as result of atresia of the pulmonary valve and the associated high pressure in the right ventricle during systole.

Paul, Sweet, and White [14] have reviewed the case of a young boy who demonstrated a continuous murmur, heard over the sternum but best located at the lower right border of the sternum, arousing suspicion of an arteriovenous fistula with possible involvement of the coronary vessels. This was confirmed at operation as the right coronary artery was found to be connected with a large venous plexus. No attempt was made to divide the arteriovenous fistula or ligate the vessel for fear of damaging the circulation. In the same paper the authors refer to a 16-year-old boy operated upon by Gross. The preoperative diagnosis was a patent ductus arteriosus

complicated by Streptococcus bacterial endarteritis. A harsh continuous murmur with a thrill was present to the left of the sternum in the third and fourth interspaces. At operation, an abnormal area, characterized by bulging and thinning of the myocardium, was found in the lateral wall of the left ventricle. No definitive surgery was undertaken. In this case the postoperative diagnosis was an arteriovenous aneurysm, probably of the coronary system.

In 1918, Blakeway and, in 1922, Reed, each described hearts in which there was direct communication between the chambers of the left ventricle and the left coronary artery.

In Case 4, although an atrial septal defect was considered in the diagnosis, the presence of a patent ductus arteriosus was not ruled out in view of the continuous character of the murmur, the high pulse pressure, and the electrocardiographic changes. The autopsy findings were very illuminating. A left-to-right shunt was produced by an intracardiac defect, presumably congenital in origin, namely, a rupture of an aortic sinus aneurysm into the right atrium. Taussig [15] has stated that aneurysms of the sinus of Valsalva almost invariably rupture into the right heart and are easily differentiated from an aortic septal defect.

In 1950, Morgan and Burchell [16] reported the case of a 34-year-old white man who presented the clinical findings of a patent ductus arteriosus, yet catheterization data suggested a left-to-right shunt proximal to the pulmonary valve. An earlier diagnosis of a ventricular septal defect was later confirmed at autopsy; however, in addition to the septal defect, there was found an aortic sinus aneurysm, with multiple perforations that communicated with the right ventricle. The presence of the continuous murmur was thought to be related to the perforated sinus aneurysm. Another somewhat similar case concerned a 13-month-old boy, reported by Brown and Burnett,[17] whose clinical examination suggested a large patent ductus. At autopsy an anomalous channel was found arising beside a normal left coronary artery in the left posterior sinus, passing down into the right ventricle along the lower border of the pulmonary conus. No surgery was undertaken. Death was due to upper respiratory infection.

The subject of aortic sinus aneurysms has been reviewed by Jones and Langley,[18] who presented the case of a 12-year-old boy with a congenital aneurysm of the right coronary sinus associated with a high ventricular septal defect. These malformations clinically simulated a patent ductus arteriosus. In a review of the same subject Venning [19] emphasized that the correct diagnosis of aortic sinus aneurysms is unlikely to be made during life unless there is rupture of one of these into the chambers of the heart. He added that the diagnosis depended upon the alteration in hemodynamics which produces a machinery-like murmur. White and others have indicated that the rupture of a syphilitic aneurysm into the pulmonary artery may produce a typical machinery-like murmur. In these cases there is usually a history of a sudden onset of cardiac symptoms, often with rapidly progressing heart failure. Schattenberg and Harris [20] stated that 3% of all ruptured syphilitic aortic aneurysms open into the pulmonary artery. The Kahn test is usually positive.

Recently, Brown, Heath, and Whitaker [21] reported the case of a 17-year-old girl who had been referred to them with a history of a known cardiac murmur since the age of 9 years and with a diagnosis of a patent ductus arteriosus on the basis of a continuous left parasternal murmur. Their complete investigation indicated a left-to-right shunt from the aorta into the right ventricle, presumably from a ruptured congenital aneurysm of a sinus of Valsalva. Open cardiotomy under hypothermia resulted in the complete closure of the fistula. However, during the procedure cardiac dilatation and fibrillation occurred. Normal functional rhythm was restored. Bacterial endocarditis and an uncontrolled Staphylococcus infection complicated the illness, and the patient succumbed on the 14th postoperative day. These authors are of the opinion that, although the

signs observed in this case are characteristic of a cardioaortic fistula, they are not pathognomonic of one, since identical signs occur in the much commoner anomalies of patent ductus arteriosus and aortic-pulmonary septal defect.

SUMMARY

There are certain congenital malformations of the cardiovascular system which may imitate the patent ductus arteriosus. Some of these lesions have been described and should be considered in patients with continuous heart murmur.

The case of a 4-month-old infant is described in whom an aortic-pulmonary fistula was suspected, but a patent ductus arteriosus could not be excluded. At operation, a ligamentum arteriosum was found. The congenital communication between the aorta and pulmonary artery was closed successfully.

The problems associated with a persistent continuous murmur in a 3-year-old infant are reviewed. A patent ductus arteriosus was found on the left to account for the continuous murmur there. A second thoracotomy ruled out a right-sided patent ductus, but the possibility of a small aortic-pulmonary fistula or an intracardiac arteriovenous fistula associated with the demonstrated interventricular septal defect has not been excluded.

A case is presented of a 19-year-old woman who, without symptoms, had the findings of a continuous machinery-like murmur, imitating a patent ductus arteriosus. At operation, no patent ductus was found; however, an anomalous left coronary artery was identified and found to shunt blood into the infundibulum of the right ventricle. Complete division of this vessel resulted in correction of the arteriovenous "fistula." No conduction changes referable to the procedure occurred.

The problems are discussed in the case of a 43-year-old man who was referred for a mitral commissurotomy. From the history and the actual findings, both physical and laboratory, the preoperative impression included the following considerations: atrial septal defect, aberrant pulmonary veins, and aortic insufficiency. And patent ductus arteriosus was considered because of the continuous murmur, wide pulse pressure, and the electrocardiographic changes of right ventricular hypertrophy. The catheterization data tended to support the diagnosis of atrial septal defect, but the murmurs were not consistent with this diagnosis. Surgical exploration of the right side of the heart suggested a high-lying interventricular septal defect with mild involvement of the atrial septum. It is possible that with open cardiotomy under hypothermia the lesion found at autopsy might have been corrected. The continuous murmur is explained by the presence of a ruptured aortic aneurysm of the right sinus of Valsalva which communicated with the right atrium. This fistula accounted for the high oxygen content in the right atrium and the increased pulmonary flow demonstrated preoperatively.

Catheterization studies, angiocardiography, and retrograde aortography are most helpful in distinguishing these lesions from a patent ductus arteriosus.

If, at operation, a suspected patent ductus arteriosus is not found, one should not fail to search for some other pathological condition, such as one of the lesions mentioned above, to account for the continuous systolic and diastolic murmurs.

REFERENCES

1. Gasul, B. M.; Fell, E. H., and Casas, R.: Diagnosis of Aortic Septal Defect by Retrograde Aortography: Report of a Case, Circulation **4**:251, 1951.

2. Dadds, J. H., and Hoyle, C.: Congenital Aortic Septal Defect, Brit. Heart J. **11**:390, 1949.

3. Gibson, S.; Potts, W. M., and Langewisch, W. H.: Aortic-Pulmonary Communication Due to Localized Congenital Defect of the Aortic Septum, Pediatrics **6**:357, 1950.

4. Holman, E.; Gerbode, F., and Purdy, A.: The Patent Ductus: Review of Seventy-Five Cases with Surgical Treatment Including Aneurysm of Ductus and One of Pulmonary Artery, Thoracic Surg. **25**:111, 1953.

5. Spencer, H., and Dworken, H. J.: Congenital Aortic Septal Defect with Communication Between Aorta and Pulmonary Artery: Case Report and Review of Literature, Circulation **2**:880, 1950.

6. Scott, H. W., Jr., and Sabiston, D. C., Jr.: Surgical Treatment For Congenital Aorticopulmonary Fistula: Experimental and Clinical Aspects, J. Thoracic Surg. **25**:26, 1953.

7. Kelsey, J. R., Jr.; Gilmore, C. E., and Edwards, J. E.: Bilateral Ductus Arteriosus Representing Persistence of Each Sixth Aortic Arch: Report of a Case in Which There Were Associated Isolated Dextrocardia and Ventricular Septal Defects, A. M. A. Arch. Path. **55**:154, 1953.

8. Dammann, J. F., Jr., and Sell, C. G. R.: Patent Ductus Arteriosus in Absence of Continuous Murmur, Circulation **6**:110, 1952.

9. Gasul, B. M.; Fell, E. H.; Dillon, R., and Marienfeld, L. H.: Diagnosis and Treatment of the "Malignant" Type of Patent Ductus Arteriosus in Infants: Clinical, Angiocardiographic and Cardiophysiologic Study of 12 Patients, Transactions of The Annual Meeting of the American Heart Association, Chicago, 1954.

10. Davis, C., Jr.; Dillon, R.; Fell, E. H., and Gasul, B. M.: Anomalous Coronary Artery Simulating Patent Ductus Arteriosus, J. A. M. A. **160**: 1047, 1956.

11. White, P. D.: Heart Disease, New York, The Macmillan Company, 1951, Ed. 4, pp. 343, 527.

12. Williams, R. R.; Kent, G. B., Jr., and Edwards, J. E.: Anomalous Cardiac Blood Vessel Communicating with the Right Ventricle: Observations in a Case of Pulmonary Atresia with Intact Ventricular Septum, A. M. A. Arch. Path. **52**:480, 1951.

13. Alexander, W. S., and Green, H. C.: Coronary Blood Vessel Arising from Cardiac Ventricle: Report of a Case Showing Other Cardiac Anomalies, A. M. A. Arch. Path. **53**:187, 1952.

14. Paul, O.; Sweet, R. H., and White, P. D.: Coronary Arteriovenous Fistula: Case Report, Am. Heart J. **37**:441, 1949.

15. Taussig, H. B.: Congenital Malformations of the Heart, New York, Oxford University Press, 1947, pp. 344, 418.

16. Morgan, E. H., and Burchell, H. B.: Symposium on Cardiac Catheterization: Ventricular Septal Defect Simulating Patent Ductus Arteriosus, Proc. Staff Meet. Mayo Clin. **25**:69, 1950.

17. Brown, R. C., and Burnett, J. D.: Anomalous Channel Between Aorta and Right Ventricle, Pediatrics **3**:597, 1954.

18. Jones, A. M., and Langley, F. A.: Aortic Sinus Aneurysms, Brit. Heart J. **11**:325, 1949.

19. Venning, G. R.: Aneurysms of Sinuses of Valsalva, Am. Heart J. **42**:57, 1951.

20. Schattenberg, H. J., and Harris, W. H.: Aortic Aneurysm with Rupture into the Pulmonary Artery, Am. Heart J. **25**:512, 1943.

21. Brown, J. W.; Heath, D., and Whitaker, W.: Cardioaortic Fistula: A Case Diagnosed in Life and Treated Surgically, Circulation **12**:819, 1955.

Atypical Patent Ductus

Results of Surgical Treatment in Infants, Patients with Pulmonary Hypertension, and Patients with Coarctation of the Aorta

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The indications for operating upon a patent ductus arteriosus have now become so well established that virtually all groups recommend closure after 4 or 5 years of age, regardless of whether the ductus is causing symptoms. We are inclined to recommend operation after 3 years of age, and we have not refused operation to any patient on the basis of age.

In recent years an increasing number of patients have been found whose clinical picture differs from the classical.* Many of these are infants who develop heart failure or severe symptoms, children or adults who have an associated pulmonary hypertension, and those who have associated cardiac or great-vessel abnormalities. It is the purpose of this paper to report our experiences in treating 18 infants less than three years of age who had serious symptoms from patent ductus, 11 children and adults who had pulmonary hypertension in association with patent ductus and 7 patients who had coarctation of the aorta and patent ductus among a total of 170 patients who were operated upon for patent ductus arteriosus (Table 1). Two patients with coarctation and patent ductus were infants of 2 weeks and 8 weeks of age, thus bringing the number of infants under 3 who were operated upon for patent ductus to 20. The first 75 of this series have been previously reported.* There were two postoperative deaths in the entire group. One patient, our fifth, died of complications resulting from shock and massive blood replacement. The other fatality occurred in an infant of 2 weeks who had multiple cardiac anomalies — atresia of the aortic arch, coarctation, and a patent ductus. This patient died from heart failure and shock.

The 20 infants were operated upon for an open ductus before 3 years of age because of

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* References 1, 4, 9, 12, and 16.

TABLE 1.—*Patent Ductus Arteriosus*

Cases with Operation: Age and Sex Distribution

	Under 3	3-5	6-10	11-15	16-20	21-25	26-30	31-35	36-40	41-45	Total
Male	6	17	13	5	3	2	..	3	2	0	51
Female	14	27	40	15	12	3	4	4	0	0	119
Total	20	44	53	20	15	5	4	7	2	0	170
Ratio: female to male	2:1	1.6:1	3:1	3:1	4:1	1:1	4:0	1.3:1	0:2	0	2.3:1

heart failure, repeated severe respiratory infections, severe dyspnea, or failure to grow, their symptoms frequently having appeared soon after birth. A feature of several patients was episodes of atelectasis caused by cardiomegaly. An example of this complication is Patient 145, whose greatly enlarged left atrium caused left-upper-lobe atelectasis and an almost fatal issue on two occasions (Fig. 1).

The characteristic signs of patent ductus are usually not found at this age, making the diagnosis difficult at times. Often the murmur is systolic only, due to the absence of a

Fig. 1.—Preoperative roentgenogram on Patient 145 showing the marked cardiac enlargement which was responsible for the recurrent episodes of left upper-lobe atelectasis.

gradient of pressure between the systemic and pulmonary circulations caused by the increased pulmonary vascular resistance. The murmur is found over the second to fourth interspace, there is a split or loud P_2, and there may or may not be a diastolic murmur at the second left interspace or apex. Thus, of 20 patients in this group, 10 had only a systolic murmur in the pulmonic area, and in 3 questionable diastolic murmurs were found. Although both systolic and diastolic, or continuous, murmurs were heard by most observers in the remaining patients, they often lacked the characteristic machinery quality associated with a ductus in children and adults. In the presence of congestive failure it was often impossible to hear a murmur at all. The blood pressures frequently were difficult to obtain accurately, but it will be noted that a wide pulse pressure was often absent.

Another characteristic feature of the young infants was the presence of a right ventricular hypertrophy pattern in the electrocardiogram, which can be considered to be normal for this age, and in those nearing 3 years of age the electrocardiogram tended to become normal. A normal electrocardiogram in the small infant might be considered as being indirect evidence of left ventricular hypertrophy. Roentgenograms and fluoroscopy showed generalized cardiac enlargement, increased vascular markings, and enlargement of the pulmonary artery, all suggesting a left-to-right shunt of some type, though not being diagnostic of patent ductus. The left atrium was commonly enlarged, as shown by the double contour and displacement of the esophagus to the right in the anteroposterior projections and by posterior displacement of the barium-filled esophagus in the right anterior oblique projections. Both ventricles, or, more commonly, the left ventricle, was enlarged. In six instances a retrograde brachial aortogram was used as a diagnostic aid and was of great help in establishing the diagnosis.† Cardiac catheterization was only used once to confirm the diagnosis in these 20 patients. Associated anomalies contributed to the poor circulatory status in at least five instances.

Pulmonary-artery pressures were measured during open thoracotomy in 7 of the 20 infants, and the highest mean pressure recorded with an open ductus was 77 mm. Hg and the lowest 30 mm. Hg (Table 2). In general, this group had more pulmonary hypertension than would be found at the later age. It will be noted, for example, that in five patients in whom the pressures were recorded there was not only a higher than

† References 3, 11.

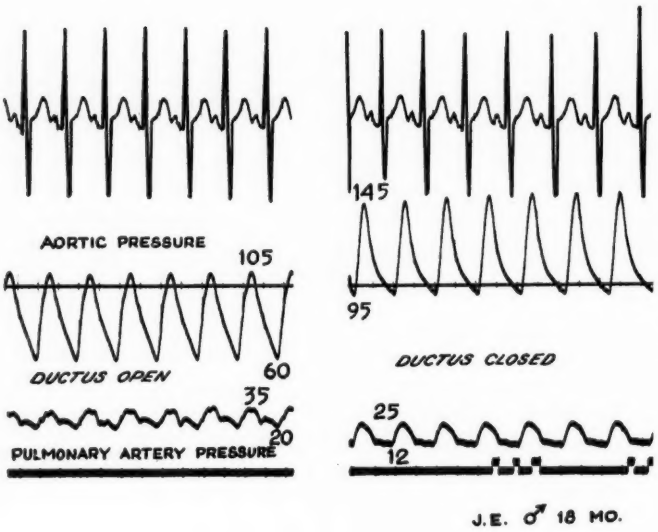

Fig. 2.—Normal change in dynamics when a patent ductus is closed. Tracings of pulmonary artery and aortic pressures taken during operation on Patient 168. Notice the sharp rise in systemic and fall in pulmonic pressures on closure of the ductus. It is customary for bradycardia to occur also.

normal pressure with the ductus open but there was also some residual pulmonary hypertension with the ductus closed. It is well known, of course, that at this age a somewhat higher pulmonary-artery pressure is to be expected under normal conditions. In six patients lung biopsy specimens were obtained, but only in one was there evidence of thickening in the pulmonary arteries (Patient 145). Characteristically, these were sickly infants who fed poorly, were subject to repeated respiratory infections, and had a loud systolic murmur in the pulmonic area and cardiac enlargement. In each instance closure of the ductus resulted in improvement or cure.

PATENT DUCTUS WITH PULMONARY HYPERTENSION

There were 11 patients more than three years of age with an open ductus and pulmonary hypertension (Table 3). The most frequent symptoms found among this group were fatigue, severe respiratory infections, dyspnea, and poor appetite. There was usually a loud systolic murmur in the pulmonic area, which occasionally carried over into diastole. Cardiac enlargement was invariably present. Again, as in the infants who had severe symptoms with ductus, the electrocardiogram in most instances showed a right ventricular hypertrophy pattern. Roentgenograms showed cardiomegaly, a large pulmonary artery, and generally increased pulmonary vascular markings.

The degree of pulmonary hypertension varied between a high of 117/70 mm. Hg in one patient and a low of 32 mm. Hg in another, as measured through open thoracotomy or by cardiac catheter. In four patients the pressures in the two systems were so close to being equal as to classify the ductus as being balanced. In two of these four patients there was definite evidence of pulmonary atherosclerosis shown in the lung biopsy specimen. Although there is still diagnostic evidence of pulmonary hypertension in these four patients with a balanced ductus, there has been clinical improvement in all.

In only 2 of the 11 patients with open ductus and pulmonary hypertension did the pulmonary-artery pressure return to normal immediately after closure (Figs. 2 and 3). In the remaining 9 patients it must be presumed that there was a factor of increased pulmonary vascular resistance, although

TABLE 2.—*Patent Ductus Arteriosus in Infancy*

Patient Number	Age	Sex	Investigations		Preop. B.P., Right	P.A. Pressures at Operation, Mm. Hg		Lung Biopsy	Result	Murmurs	Reasons for Operation	Remarks
			E.C.G.	X-Ray		Ductus Oper	Ductus Closed					
121	5 mo.	M	R.V.H.*	Enlarged heart; large P.A.§; L.V. & P.A. enlarged; retrograde aortogram: P.D.A.‖	110/0	Large tense P.A.		.....	Improved	Systolic	Dyspnea; pneumonia; respiratory infections; failure to gain	Postop. roentgenograms suggest pulmonary hypertension plus perhaps an associated septal defect
112	6 mo.	F	R.V.H.	Enlarged heart; prominent vascular markings; retrograde aortogram: P.D.A. plus other anomalies	118/40	Attempted		.....	Much improved	Systolic	Dyspnea; orthopnea; cyanosis; heart failure	Systolic murmur on discharge
138	7 mo.	M	Normal	Enlarged heart; enlarged right ventricle	140	..	..	.....	Much improved	Systolic	Fatigue and failure to gain; rapid respirations	.....
123	8 mo.	F	R.V.H.	Enlarged R ventricle & L auricle; P.A. very large; retrograde aortogram: P.D.A.	110/20	45	32	Normal	Much improved	Systolic	Frequent respiratory infections; dyspnea; slow growth; cardiomegaly	Possible interventricular septal defect
130	13 mo.	F	L & R ventricular hypertrophy	Huge P.A.; large L atrium and biventricular enlargement; retrograde aortogram: P.D.A.	105/70	44	43	Chronic hyperemia	Much improved	Systolic	Cardiomegaly; intermittent vomiting	Still has murmur to the left of sternum
145	14 mo.	M	R.V.H.	Enlarged heart; prominent pulmonary vessels	70/10	33	33	Sclerosis	Much improved	Systolic and diastolic	Severe respiratory infections; marked cardiomegaly causing atelectasis	.....
153	14 mo.	F	R.V.H.	Large P.A.; R ventricular and atrial enlargement; retrograde aortogram: P.D.A.	80/50	77	64	.....	Much improved	Systolic	Heart failure	Possible associated interatrial septal defect
168	18 mo.	M	R.V.H., including R.B.B.B.†	Enlarged heart; prominent pulmonary vessels	110/60	35/20	25/12	Normal	Cured	Systolic and diastolic	Severe respiratory infections; dyspnea and fatigue	.....
70	18 mo.	M	Normal	Very large heart; P.A. enlarged	90/60	P.A. tense		.....	Cured	Systolic	Cardiomegaly; heart failure; repeated severe respiratory infections	.....

TABLE 2.—*Patent Ductus Arteriosus in Infancy.* (Continued)

Patient Number	Age	Sex	Investigations		Preop. B.P., Right	P.A. Pressures at Operation, Mm. Hg		Lung Biopsy	Result	Murmurs	Reasons for Operation	Remarks
			E.C.G.	X-Ray		Ductus Open	Ductus Closed					
112	20 mo.	F	R.V.H.	Marked cardiomegaly R & L ventricular hypertrophy; prominent P.A.; retrograde aortogram: P.D.A.	80/60	Taken but not recorded; normal after closure		Normal	Cured	Systolic	Severe respiratory infections; failure to gain; dyspnea; marked cardiomegaly	.....
163	20 mo.	F	Latent A.V.‡ block; left axis deviation	Enlarged heart; enlarged L.A. & L.V.; prominent P.A.	138/70 (crying)	..	..	.....	Improved	Systolic and diastolic	Failure to gain weight; respiratory infections; cardiomegaly	.....
90	2 yr.	F	Normal	L ventricle large, also R ventricle	124/40	..	..	.....	Cured	Systolic and diastolic	Bronchopneumonia; repeated severe respiratory infections	.....
71	2 yr.	F	R.V.H.	Very large heart; L ventricular enlargement	100/50	30	29	.....	Improved	Systolic and diastolic	Slight dyspnea; poor eater	Other congenital abnormalities; possible residual pulmonary stenosis or interventricular septal defect
74	2 yr.	F	Normal	Enlarged heart; L.V.H.¶	110/40	..	..	.....	Cured	Systolic	Failure to grow; respiratory infections	.....
53	2 yr.	F	Normal	L.V.H.; moderately enlarged L atrium	110/60	..	..	.....	Much improved	Systolic; occasionally diastolic	Shortness of breath	Thought to have enlarged thymus; ? other cardiac defect; systolic murmur still heard 4 mo. after operation
81	2½ yr.	F	Normal	L ventricular enlargement (minimal)	120/50	..	..	.....	Cured	Continuous	Failure to gain; frequent respiratory infections	.....
84	2½ yr.	F	.....	Cardiac enlargement	112/80	..	..	.....	Cured	Continuous	Frequent respiratory and urinary infections; dyspnea	.....
158	2½ yr.	M	Early L.V.H. aberrant R.V. conduction	L ventricular and cardiac enlargement	102/60	..	..	.....	Cured	Continuous	Cardiomegaly	.....

* Right ventricular hyptertrophy.
† Right bundle branch block.
‡ Atrioventricular.
§ Pulmonary artery.
‖ Patent ductus arteriosus.
¶ Left ventricular hypertrophy.

TABLE 3.—*Pulmonary Hypertension*

Patient Number	Age, Yr.	Sex	Cardiac Catheter Pressures, Mm. Hg	X-Ray	E.C.G.	Preop. B.P., Right	P.A. Pressures, Mm. Hg		Lung Biopsy	Result	Murmurs	Symptoms	Remarks
							Ductus Open	Ductus Closed					
118	3½	M	.....	L.V.§ enlargement; prominent pulmonary vessels	.....	100/55	36	31	No diagnosis	Cured	Continuous	Dyspnea and fatigue	.....
125	3½	F	.....	Cardiac enlargement R. & L.V.; L.A.‖ & P.A. enlarged; retrograde aortogram: P.D.A.¶	Right axis deviation	90/70	65/35	60/30	Interstitial pneumonitis	Much improved	Systolic	Heart failure; repeated severe respiratory infections; dyspnea	Mild aortic stenosis
98	4	M	Aorta 70/43; R.V.* 74/0; P.A.† 72/40; arterial O_2-87%	Enlarged heart due mainly to R.V.; large P.A.; engorged vessels	R.V.H.≠	100/50	Not measured; P.A. very tense		No diagnosis	Improved	Systolic; occasionally diastolic	Frequent respiratory infections	Still has Graham Steell murmur and loud P_2
129	5	F	.....	L.A. enlargement; P.A. full	Normal	104/55	32	24	Normal	Cured	Continuous	Asymptomatic	.....
154	6½	F	Aorta 82/53; R.V. 88/8; P.A. 87/57; O_2 saturation 89%	Cardiomegaly; biventricular enlargement, including pulmonary vascularity; retrograde aortogram negative	R.V.H.	110/60	115/70	110/70	Normal	Improved	Systolic	Increasing dyspnea	Loud P_2 remains
117	8	M	P.A. 79/55; R.V. 79/2; F.A.‡ 97/64; aorta through ductus 79/59; postop. normal; O_2 saturation R toe & R finger 97%	Markedly enlarged heart; large P.A.; large R.A. & V.	Normal	110/80	55	35	Normal	Much improved	Systolic	Failure to grow; frequent severe respiratory infections; dyspnea and fatigue	Loud P_2 remains
107	10	F	R.V. 69/4; P.A. 66/42; F.A. 122/50; O_2 saturation 94%	Cardiomegaly; R.V. enlarged, L.V. slightly enlarged; P.A. markings prominent	Normal	106/56	60	20	No diagnosis	Cured	Loud systolic; short diastolic	Poor growth; frequent respiratory infections; fatigue	.....
142	11	F	R.V. 96/7; P.A. 99/64; O_2 saturation finger & toe 92%	Cardiac enlargement due to R.V. & L.A.; pulmonary vascular engorgement	R.V.H.	120/70	80	60	Normal	Died 9 mo. postop.	Systolic and diastolic	Poor growth; repeated attacks of pneumonia; severe dyspnea	Death due to pneumonia
143	11	M	R.V. 85/7; P.A. 86/61; F.A. oxygen saturation 89%	Enlarged heart; prominent P.A.	R.V.H.	100/70	86	74	Extensive arteriosclerosis thrombosis & recanalization	Improved	Systolic	Slow growth; fatigue; severe respiratory infections; dyspnea	Multiple congenital abnormalities; loud P_2 remains
110	16	F	R.V. 117/11; P.A. 109/79; aorta 110/75; descending aorta O_2 saturation 75%	R.V. enlargement; large P.A.	R.V.H.	120/80	75	100	Phlebosclerosis & arteriosclerosis	Unimproved; ductus not closed	Systolic	Fatigue; dyspnea; cyanosis of feet	No closure because of increase in P.A. pressure on occlusion of ductus
85	33	F	R.V. 100/10; P.A. 100/62; P.A. 136/60; brachial A. 90%; F.A. 87%; 5 wk. postop.: R.V. 65/4, P.A. 65/4	Enlarged heart; R. & L.V. & possibly L.A. enlargement; P.A. large; engorged vessels	R. & L.V.H.	130/60	70/60	60/50	Moderate arteriosclerosis thrombosis & recanalization	Much improved	Systolic; very occasionally diastolic	Mild heart failure	Nearly asymptomatic; loud P_2 remains

* Right ventricle.
† Pulmonary artery.
‡ Femoral artery.
§ Left ventricle.
‖ Left atrium.
¶ Patent ductus arteriosus.
≠ Right ventricular hypertrophy.

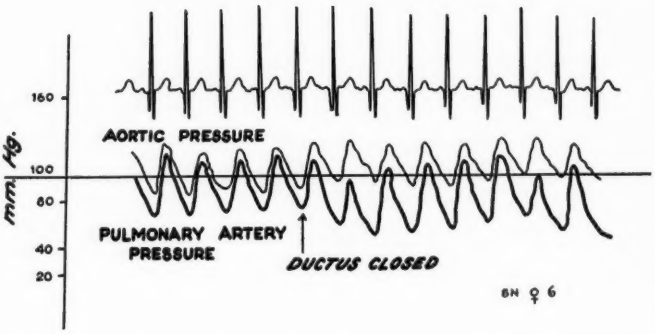

Fig. 3.—Tracings of pulmonary artery and aortic pressures recorded during operation on Patient 154, a 6½-year-old girl with a residual pulmonary artery pressure of approximately 110/70 mm. Hg after closure of the ductus. Notice that in the presence of marked pulmonary hypertension the systemic pressure changes little when the ductus is closed.

pathologic evidence of vascular disease, as seen in lung biopsy specimens obtained at operation from the lingula, showed definite changes in only three of the entire group. Here, however, the changes were uniformly extensive and consisted of atherosclerosis, thrombosis, and recanalization, all indications of a process which had existed for a long time, perhaps from birth. The youngest of these patients was Patient 143, who was an 11-year-old boy with multiple congenital abnormalities and a pulmonary artery pressure of 86 mm. Hg, which fell to 74 mm. Hg after closure (Fig. 4).

The factors which account for residual pulmonary hypertension in the presence of normal-appearing vessels are poorly understood.‡ It should be remembered that pathological examination of a lung biopsy specimen is a poor estimate of the amount of blood these vessels might carry under various circumstances, for the element of physiologic response in the living state is lost in the biopsy. Furthermore, we are only assessing the structural changes of the vessels in the excised segment.

One patient in this group had definite and continuous reversal of flow through the ductus. This was a 16-year-old girl who was moderately incapacitated from the defect (Patient 110). Upon physical examination there was blueness and clubbing of the toes and normal-appearing fingers. Cardiac catheterization confirmed the diagnosis. At operation a large ductus and a very tense distended pulmonary artery were found. The pressure in the pulmonary artery rose from 75 mm. Hg to 100 mm. Hg with temporary closure of the ductus, and the pulmonary artery be-

‡ References 1, 2, 4, 5, 9, 14, and 15.

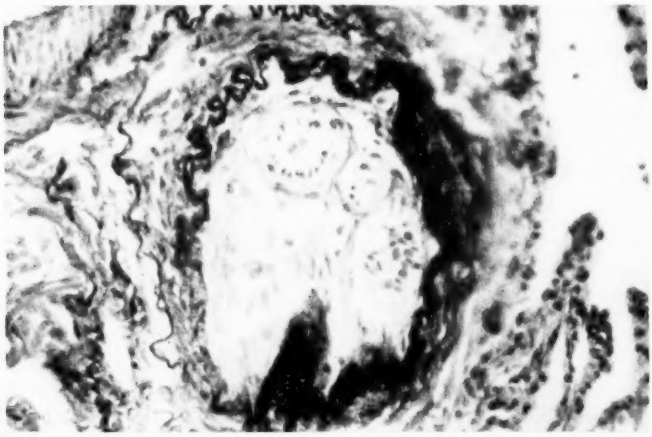

Fig. 4. — Photomicrograph of a section of lingula removed at operation from Patient 143, an 11-year-old boy with patent ductus and a residual pulmonary hypertension of 74 mm. Hg after closure of the ductus. This section shows recanalization of a thrombosed pulmonary artery with three new vessels. It also shows thickening of the artery wall and fragmentation of the elastica.

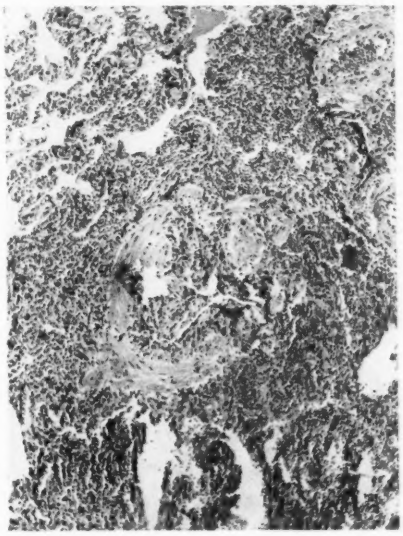

Fig. 5.—Photomicrograph of a section of lingula removed at operation from Patient 110, a girl of 16 with a reverse ductus. The section shows a pulmonary vessel with a lumen which is filled with fibrous tissue in which there are two small vascular spaces representing recanalization following an organized thrombus.

came larger. At the same time, the heart enlarged and the rate slowed. We therefore desisted from permanent closure, for a fatal issue would have resulted. This girl has continued to be somewhat dyspneic and has fatigue, but she can manage light housekeeping. The lung biopsy specimen showed rather marked atherosclerosis and thrombosis involving the small pulmonary arteries (Fig. 5).

The only patient in this group who is not living was an 11-year-old girl (Patient 142) who had a pulmonary-artery pressure of 99/64 mm. Hg preoperatively, 80 mm. Hg in the operating room with the ductus open, and 60 mm. Hg with it closed. Her condition was unchanged by closure of the ductus, for she continued to have dyspnea, failure to gain, and fatigue, and the physical signs continued to be those of pulmonary hypertension and right ventricular strain nine months after the operation. We had planned to study her again, but she died of pneumonia, which was largely caused by the extremely poor conditions under which she had to live. It is of interest to note that the lung biopsy specimen was normal in this case. The remaining nine patients with pulmonary hypertension who had the ductus closed are improved or cured, although most of them continue to have evidence of pulmonary hypertension.

PATENT DUCTUS WITH COARCTATION OF THE AORTA

Among the 170 patients with patent ductus, there were 7 who were operated upon for combined patent ductus and coarctation. (Table 4 and Fig. 6) The youngest of this group was a 2-week-old infant who had been in heart failure since birth. As a desperate measure operation was performed. A narrow aortic arch and a coarctation with a large ductus emptying into the distal aorta were found. The repair was not difficult, but the infant died during the first postoperative day. A second infant with severe heart failure due to coarctation plus a ductus which entered the distal aorta was operated upon at eight weeks. She has made excellent progress and looks like a normal child, although there may still be a small septal defect present.[6] In two patients of this group the ductus virtually entered the aorta at the coarctation, although they should probably be classified as entering proximally. Of the remaining three patients, it was clear that in one the ductus entered proximally and in two it entered distally.

The clinical findings in these patients differed in several respects from straightforward ductus or coarctation. They had more symptoms, such as dyspnea, respiratory infections, and heart failure. All save the infant of two weeks had hypertension in the upper extremities, but they also tended to sustain a fairly good, though lower, pressure in the legs. All had systolic murmurs, and most, in addition, had the characteristic basal diastolic murmur usually found in the combined lesion. The diagnosis was made or suspected on routine studies, with one exception where cardiac catheterization was used. Both lesions were repaired in one operation,

TABLE 4.—*Coarctation of the Aorta and Patent Ductus Arteriosus*

Patient Number	Age	Sex	Cardiac Catheterization Pressure, Mm. Hg	E.C.G.	X-Ray	Preop. B.P., Right	P.A. Pressures, Mm. Hg. Ductus Open	P.A. Pressures, Mm. Hg. Ductus Closed	Lung Biopsy	Result	Murmurs	Reasons for Operating	Remarks
120	2 wk.	M	.....	R.V.H.§	Very large heart; congested lungs; large P.A.	A 60 L 66	..	..	Normal	Died	Initially systolic then diastolic	Heart failure; increasing cyanosis; severe dyspnea	Died postop.; ?? intracranial lesion
99	8 wk.	F	.....	A. & V.H.‖	Huge heart; R.A. & V.H.; pulmonary vessels engorged; some atelectasis	A 140 L 40	..	..	.....	Much improved	Systolic	Heart failure; pneumonia; atelectasis; severe dyspnea	Hypothermia; ductus distal to coaretation; questionable interventricular septal defect
164	6 yr.	F	.....	.....	Left ventricular enlargement	A 132/47 L 67/70	..	24	.....	Much improved	Continuous	Nose bleeds; dyspnea	At operation, marked poststenotic dilatation
149	7 yr.	M	R.V.* 76/9; P.A.† 79/38; F.A.‡ 87/38	R. & L.V.H.	Enlarged heart; both ventricles & left atrium enlarged; peripheral pulmonary vessels not as prominent as central	A 190/70 L 80/60	..	..	.....	Improved	Systolic and diastolic	Hypertension	Ductus opposite coaretation
137	7 yr.	F	.....	R.V.H.	Rib notching; enlarged left ventricle; some enlargement R.V.; left atrium enlarged; question of abnormal venous return	A 170/30 L 110/84	55	55	Hyperaemia	Cured	Systolic and diastolic	Hypertension	Ductus distal to coaretation
33	10 yr.	F	.....	R.A.D.¶	R. & L. ventricular enlargement; large P.A.; lungs engorged	A 155/40 L 115/50	..	..	.....	Improved	Continuous	Mild failure; fatigue; attacks of cyanosis	Coaretation excised at second operation
115	10 yr.	F	.. ..	Normal	Enlarged heart; primary left atrium and ventricle; prominent vascular markings	A 142/74 L 140/76	.	..	.....	Cured	Systolic and diastolic	.....	Ductus distal to coaretation

* Right ventricle.
† Pulmonary artery.
‡ Femoral artery.
§ Right ventricular hypertrophy.
‖ Atrial and ventricular hypertrophy.
¶ Right axis deviation.

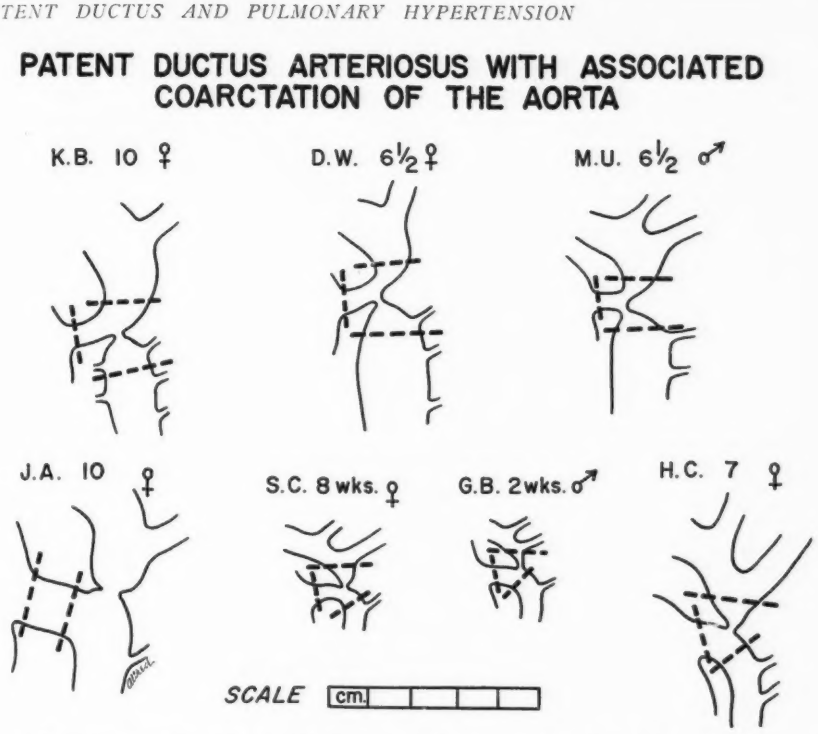

Fig. 6.—Drawings of the local anatomy made at operation. The dotted lines show the areas of transection. The drawings are accurately made as to size of the involved vessels.

except Patient 33 in whom it was thought advisable to close the ductus first and repair the coarctation later. Pulmonary artery pressures were measured in two patients with pulmonary hypertension. In one there was a slight reduction in pressure after repair, and in the other there was no change.

The six surviving patients were improved or cured by operation. They all lead active lives and no longer have dyspnea or repeated respiratory infections. The blood pressures have reached normal in all patients except one (Patient 33) where there is still a mild elevation in the arm pressures.

PREOPERATIVE CARE OF THE INFANT WITH PATENT DUCTUS ARTERIOSUS

Preoperative care, and actually prediagnostic care, is of the utmost importance. Most infants with patent ductus arteriosus have eaten poorly and may have subsisted on milk alone, which is an inadequate diet after the first few weeks of life, even if some vitamin product is added. This may give rise to an iron-deficiency anemia, which may aggravate the symptoms of the cardiac defect and increase the risk of surgery. Treatment is either by iron medication if there is time or by blood transfusions if more rapid correction of the anemia is desired.

The two other complications which require treatment are cardiac failure and pneumonia, and these are so often a part of the picture of the infant with a large ductus that they frequently are the conditions which focus the attention of the physician on the cardiac defect. These two conditions are often present together, and it is difficult to separate them in infants. Adequate antibiotic therapy, with culture of the invading organism, if possible, from the nose and throat, blood stream, or urine, is essential. In addition, proper control of the congestive failure is essential prior to surgery. The principles of treatment for

congestive failure are proper digitalization, low-salt diet, oxygen if indicated, adequate diet, and mercurial diuretics if other measures fail.

With the proper attention to these three accompaniments of the persistent ductus in infancy, surgical risk need not be higher than it is for the older child with few or no symptoms. Preparation for operation may take two weeks or more, and as a general rule surgery is postponed until conditions are optimal. On the morning of operation an intravenous drip is established on the ward through a plastic tube inserted in an ankle vein. It is far better to do this with a local anesthetic preoperatively than to utilize valuable time in the operating room. This indwelling tube is allowed to remain in place postoperatively until it is no longer needed or is causing irritation.

OPERATIVE TECHNIQUE

In most instances the ductus was exposed through a posterolateral incision through the fifth interspace. The rib was preserved, and access to the pleural space was gained through the periosteum stripped from the inferior half of the rib. Care was taken, particularly in females, not to carry the posterior extension of the incision too high in order to prevent difficulty in covering it with clothing later. The ductus was exposed by blunt and sharp dissection with displacement of the recurrent laryngeal and vagus nerves laterally. In two very sick infants where a long ductus was found, the multiple silk ligature method was used for closure to shorten the operating time. However, in the majority of the last 100 patients the ductus was divided between clamps and the ends sutured with arterial silk. Pulmonary-artery pressures were measured either by a recording electromanometer or by a saline manometer, in which case the pressures were converted into millimeters of mercury.

When there was an associated coarctation, the largest lumen possible was obtained by using an oval-shaped anastomosis and allowance for growth was made by utilizing interrupted mattress sutures for at least half of the anastomotic line. Underwater sealed drainage was used for 24 to 48 hours in all cases. Antibiotics were administered routinely for about a week postoperatively.

A very important aspect of surgical treatment within the thorax in infants is expert anesthesia. We have been fortunate in having had Dr. Philip Bailey supervise the anesthesia in most of the difficult cases in this series, and without his interest and attention it would not have been possible virtually to eliminate operative mortality.

COMMENT

Frequently, in the appraisal of an infant who is showing cardiac embarrassment the differential diagnosis is narrowed to two lesions: a persistent patent ductus arteriosus or an interventricular septal defect with a large left-to-right shunt. Theoretically, the presence of a wide pulse pressure serves to differentiate between the two and when conclusively proved is of invaluable aid. However, with the limitation in the accuracy of present-day infant cuffs, the frequent presence of a moderate degree of pulmonary hypertension in both lesions, and the difficulty of accurate blood-pressure determination, this criterion is of little practical help. The presence of a diastolic murmur is an uncertain clue, since the interventricular septal defect will frequently have a diastolic murmur over the apex. The location of the murmur is even more important. In our experience it is rare to find the maximal intensity of a systolic murmur over the fourth left interspace or xiphoid in the presence of a ductus without an associated ventricular septal defect.

Because of the ominous prognosis in both the infant with a large ductus with congestive failure and the infant with a "malignant" interventricular septal defect, these infants have been subjected to retrograde arteriography in those instances where the diagnosis of patent ductus arteriosus was in doubt, or even where there was any suspicion that a ductus was a contributing factor in the infant's symptoms and signs.

In the patients who have a patent ductus arteriosus in association with pulmonary hypertension, the clinical findings are mostly related to the degree of pulmonary vascular resistance present; this influences the amount of blood flowing through the ductus and the direction of its flow. In a common ductus there may be as much as 2 to 10 liters of blood flowing from aorta to pulmonary artery through the ductus. This causes the continuous murmur and thrill and the wide pulse pressure. As the pulmonary vascular resistance increases, owing to as yet undisclosed causes or to atherosclerotic or thrombotic occlusions in the pulmonary vascular bed, the pulmonary-artery pressure rises and the gradient between the two systems decreases. Thus we may have physiological situations where there is only a small amount of flow during diastole, and therefore only a faint diastolic murmur, or no flow during diastole, and therefore only a systolic murmur. When the pulmonary vascular resistance approximates the systemic vascular resistance there may be no appreciable flow, causing a balanced ductus. If the pulmonary vascular resistance exceeds that of the systemic, a reversed ductus with cyanosis of the distal extremities exists, or, under very severe circumstances, generalized cyanosis. As the pulmonary vascular resistance increases, its effect on ductus flow is shown by the disappearance of the wide pulse pressure.

It is not yet clear how many instances of pulmonary hypertension are caused by the wear and tear of increased pulmonary blood flow, but there is no doubt that in some the flow has at least increased the amount of vascular disease present. In some patients with patent ductus, apparent relief of the pulmonary hypertension is due to removal of the large flow passing through lungs in the presence of mild pulmonary vascular resistance. In others, closure of the ductus may effect very little immediate change in pulmonary-artery pressure, but with the passage of time there will be some improvement.

Our observations lead us to believe that when a balanced ductus exists and there is no rise in pulmonary-artery pressure with temporary occlusion of the ductus it may then be permanently closed. Clinical improvement may follow, but the signs of pulmonary hypertension usually persist in some degree. That this does not always occur is attested by the fact that one of our patients (Patient 142) showed no clinical improvement following closure and continued to have severe symptoms and signs of pulmonary hypertension; she died nine months later with a superimposed pneumonia.

If there is a rise in pulmonary-artery pressure to above the systemic mean pressure with temporary closure of the ductus or in the presence of clear-cut reversal of flow as evidenced by cyanosis of the distal extremities, closure will result in a fatal issue.

Patent ductus arteriosus in association with coarctation of the aorta may occur with the ductus opening proximal or distal to the coarctation.§ If it opens proximally it produces a profound physiological disturbance, for then a great quantity of blood is forced directly into the lesser pulmonic circulation. Collateral circulation must develop during the embryonic period to make this lesion compatible with life. When the patent ductus enters distal to the coarctation, the circulatory effect varies with the resistance in the pulmonary vascular bed and with the total pressure in the collateral bed. Thus, if the pulmonary vascular resistance is high and the collaterals poorly developed, the flow might be from the pulmonary artery into the distal aorta, in which case the lower extremities will be cyanotic. If the collaterals are well developed and the pulmonary vascular resistance is low, the flow may be toward the lungs from the distal aorta. In such cases there is no cyanosis of the feet. It is possible, of course, to have the pressures in the two systems the same, in which case there would be no flow through the ductus. In some instances, owing to local peculiarities of growth, the ductus may appear to enter

§ References 6, 10, and 13.

opposite the coarctation, in which case a combination of the various physiological disturbances results. Surgical repair of both the ductus and the coarctation is usually feasible at the same operation and results in a profoundly favorable change in hemodynamics.

SUMMARY

Variations in the signs of patent ductus may make the diagnosis difficult. This is particularly true in the patent ductus which causes heart failure in infancy and in those associated with pulmonary hypertension or coarctation of the aorta.

Among 170 patients who were operated upon for patent ductus arteriosus there were 20 infants below three years of age who had heart failure or severe symptoms; all but one were improved or cured by closure of the ductus. There were 11 children and adults with patent ductus and pulmonary hypertension; closure of the ductus was possible in 10 instances, with clinical improvement in 9 and the death of 1 nine months postoperatively. It was not possible to close a ductus with reversed flow in a 16-year-old girl. Seven patients had coarctation of the aorta and patent ductus. There was one postoperative death in this group—a two-week-old infant in heart failure with multiple cardiac defects. The others are improved or cured after repair of both anomalies.

REFERENCES

1. Burchell, H. B.: Variations in the Clinical and Pathologic Picture of Patent Ductus Arteriosus, M. Clin. North America, **32**:911-923, 1948.

2. Campbell, M.: Patent Ductus Arteriosus: Some Notes on Prognosis and on Pulmonary Hypertension, Brit. Heart J. **17**:511-533, 1955.

3. Castellanos, A., and Pereiras, R.: Retrograde or Counter-Current Aortography, Am. J. Roentgenol. **63**:559-565, 1950.

4. Damman, J. F., Jr., and Sell, C. G. R.: Patent Ductus Arteriosus in the Absence of a Continuous Murmur, Circulation **6**:110-124, 1952.

5. Edwards, J. E.; Douglas, J. M.; Burchell, H. B., and Christensen, N. A.: Pathology of the Intrapulmonary Arteries and Arterioles in Coarctation of the Aorta Associated with Patent Ductus Arteriosus, Am. Heart J. **38**:205-233, 1949.

6. Gerbode, F.; Purdy, A.; Alway, R. H.; Piel, J. J., and da Costa, I. A.: Surgical Treatment of Coarctation of the Aorta in Infancy, Am. J. Surg. **89**:1138-1143, 1955.

7. Holman, E.: Certain Types of Congenital Heart Disease Interpreted as Intracardiac Arteriovenous and Venoarterial Fistulae: Patent Ductus Arteriosus, Bull. Johns Hopkins Hosp. **36**:61-80, 1925.

8. Holman, E.; Gerbode, F., and Purdy, A.: The Patent Ductus—A Review of Seventy-Five Cases with Surgical Treatment Including an Aneurysm of the Ductus and One of the Pulmonary Artery, J. Thoracic Surg. **25**:111-142, 1953.

9. Hultgren, H.; Selzer, A.; Purdy, A.; Holman, E., and Gerbode, F.: The Syndrome of Patent Ductus Arteriosus with Pulmonary Hypertension, Circulation **8**:15-35, 1953.

10. Johnson, A. L.; Ferencz, C.; Wiglesworth, F. W., and McRae, D. L.: Coarctation of the Aorta Complicated by Patency of the Ductus Arteriosus: Physiologic Considerations in Classification of Coarctation of Aorta, Circulation **4**:242-250, 1951.

11. Keith, J. D.: Angiocardiography in Infants and Children, Pediat. Clin. North America **1**:73-92, 1954.

12. Myers, G. S.; Scannel, J. G.; Wyman, S. M.; Dimond, E. G., and Hurst, J. W.: Atypical Patent Ductus Arteriosus with Absence of the Usual Aortic-Pulmonary Pressure Gradient and of the Characteristic Murmur, Am. Heart J. **41**:819-833, 1951.

13. Taylor, B. E.; Knutson, J. R. B.; Burchell, H. B.; Daugherty, G. W., and Wood, E. H.: Symposium on Cardiac Catheterization: Patent Ductus Arteriosus Associated with Coarctation of the Aorta; Report of 2 Cases Studied Before and After Surgical Treatment, Proc. Staff Meet. Mayo Clin. **25**:62-68, 1950.

14. Welch, K. J., and Kinney, T. D.: The Effect of Patent Ductus Arteriosus and of Interauricular and Interventricular Septal Defects on the Development of Pulmonary Vascular Lesions, Am. J. Path. **24**:729-761, 1948.

15. Whitaker, W.; Heath, D., and Brown, J. W.: Patent Ductus Arteriosus with Pulmonary Hypertension, Brit. Heart J. **17**:121-137, 1955.

16. Ziegler, R.: The Importance of Patent Ductus Arteriosus in Infants, Am. Heart J. **43**:553-572, 1952.

Interatrial Septal Defects

Review of Thirty-Four Cases and Discussion of Three Current Methods of Closure

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Muller, Smith, Dammann, Adams, and Darsie [1] recently reported a series of 18 patients in whom closure of an interatrial septal defect was attempted. The Gross "well" technique was used in 15 cases and the atrioseptopexy method in 3 others. The defect was successfully closed in 14 patients, all of whom obtained good clinical results.

Sixteen of these patients are included in the present review, together with an additional 13 cases in which an attempt was made to close the defect by the atrioseptopexy technique, four cases in which closure was effected by open cardiotomy and direct suture of the defect and one case in which closure was attempted by the Gross "well" technique. While the exact incidence of defects of the atrial septum is unknown, there is good clinical evidence to suggest that this anomaly is one of the most frequent congenital cardiac defects. It has been generally considered that many patients, even with large septal defects, show few if any symptoms. Taussig [2] estimates the average duration of life for patients with interatrial septal defects as 40 years. Bailey [3] believes that too great an emphasis has been placed on the proposition that patients with uncomplicated atrial septal defects often manifest no symptoms until the third or fourth decades. Careful questioning, he believes, will bring forth evidence of disturbed cardiac dynamics in most such persons. Shortness of breath, exertional dyspnea, limitation of exercise tolerance, episodes of paroxysmal tachycardia, and chest pain were frequently present in the patients reported by him.

In the presence of atrial septal defect, the predominant shunt is from left to right, which increases the flow of blood through the right side of the heart and the pulmonary circulation. There is dilatation of the right atrium, dilatation and hypertrophy of the right ventricle, enlargement of the pulmonary artery, and engorgement of the pulmonary vascular system; the latter may be accompandied by pulmonary hypertension. The left atrium dilates only slightly, and the left ventricle remains normal in size.

Methods for closing interatrial septal defects may be generally classified into one of two groups: the closed method or the open method. A review of the current literature indicates that the various methods of "closed" repair are more widely used at the present time than the "open" method.

The atrioseptopexy method first suggested by the experimental work of Cohn,[5] and modified by Bailey,[6] has been used successfully by a number of surgeons. The Bjork-Craford technique has been successfully utilized by Cooley [7] in three cases. He states that the method appears to be an effective and relatively simple means of closing atrial defects of all types, irrespective of size, number, or location. Lam * has reported 13 patients

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* Lam, C. R.: in discussion of Lewis, Taufic, Varco, and Niazi.[4]

TABLE 1.—*Age Distribution of Cases with Operation*

Years	No. of Cases
0- 5	8
5-10	9
10-15	6
15-25	2
25-40	2
40-50	7
Total	34

who were operated upon without a death or serious complication. In the last half of his series the defect was closed by using a two-pointed needle. The "atrial well" technique proposed by Gross [8] presents certain technical difficulties which have made surgeons seek simpler methods of effecting closure.

Swan † and Lewis ‡ have advocated the "open" method, utilizing hypothermia with temperatures reduced to a point between 24.5 and 29.5 C. After a preliminary exploration of the septum with the index finger introduced through the right atrial appendage, the cardiac inflow and outflow are occluded. The right atrium is widely opened and the defect sutured under direct vision. Lewis [4] reports that the duration of inflow occlusion ranged from 3 to 7¾ minutes for 35 operations. Both Swan [10] and Lewis [4] have emphasized the anatomical variations which occur in these defects. Anomalous pulmonary venous drainage, multiple defects, and anomalous openings of venae cavae are some of the variations which may complicate closure. "Open" cardiac repair is said to offer a better opportunity for accurate closure of the defect when such conditions are encountered.

† References 9 and 10.

‡ References 4, 11, and 12.

CASE MATERIAL

Thirty-four cases in which closure of interatrial septal defect was attempted have been reviewed. All cases but one were of the ostium secundum type. There was one case with the ostium primum type of defect.

Twenty of the patients were females; fourteen were males. The age distribution of the cases with operation is shown in Table 1.

History (Table 2).—The histories have been reviewed with respect to growth and development, ability to participate in normal activities, and signs of cardiac and circulatory inadequacies.

The growth and development was normal in 19 and retarded in 13 cases. Two patients, who had normal growth, were markedly underweight.

Five patients had no limitation of their activities. Twenty-four complained of excessive fatigability. Dyspnea was present in 19 cases, and 2 patients had moderate orthopnea.

Twenty-nine patients had no history suggestive of myocardial insufficiency. One patient had mild symptoms of congestive failure; four patients had marked congestive failure. One patient complained of palpitation; another had attacks of paroxysmal tachycardia. Cyanosis was definitely present in one patient, slight in the case of two patients, questionable in two other patients, and present during episodes of congestive failure in one patient.

A history of frequent upper respiratory tract infections was elicited in 15 cases. Three patients reported attacks of pneumonia. Subacute bacterial endocarditis occurred in one patient. Vague cerebral symptoms, such as

TABLE 2.—*Histories of Thirty-Four Cases in Which Closure of Interatrial Septal Defect Was Attempted*

Growth		Activities		Congestive Failure		Cyanosis		Other Symptoms	
Normal	19	No limitation	5	None	29	None	28	Palpitation	1
Retarded	13	Excessive	5	Slight	1	Questionable	2	Paroxysmal tachycardia	1
Underweight	2	Fatigability	24	Marked	4	Slight	2	Frequent upper respiratory tract infections	15
		Dyspnea	19			Definite	1		
		Orthopnea	2			With failure	1	Pneumonia	3
								Cerebral symptoms	3
								Subacute bacterial endocarditis	1

attacks of dizziness, "blackout," and unclear thinking, occurred in three cases.

Physical Examination.—The records of the physical examinations were reviewed for indications of circulatory inadequacy. The liver was normal in size in 26 patients, slightly enlarged in 5, and considerably enlarged in 3. Edema was present in only one patient. Clubbing of the fingers was marked in one patient and slight in another. A precordial bulge was found in seven patients. Other defects noted in this series included a moderate pulmonary stenosis in one patient and blindness in one eye of another.

On physical examination the heart was normal in size in 7, slightly enlarged in 9, and definitely enlarged in 18 patients. We have considered a systolic murmur, best heard in the second or third interspace just to the left of the sternum, as a murmur that is typically associated with a patent interauricular septal defect. This type of murmur was present in 33 patients. The intensity of the murmur was loud in 25 patients and soft in 8. A diastolic murmur, generally in the same area, was present in eight patients. The murmur was considered atypical in one patient who had a loud, rough systolic murmur heard over the entire precordium, most intense just medial to the apex of the heart. A soft diastolic blow was heard in the pulmonic area.

A thrill, best felt in the second or third interspace to the left of the sternum, was present in six patients. Cardiac rhythm was normal in 31 patients. Extrasystoles, auricular fibrillation, and nodal tachycardia were each present in a single patient. The pulmonic second sound was increased in intensity in 20 patients and split in 19.

Radiological and Laboratory Examinations.—The hemoglobin was normal in 28 patients; 4 patients were slightly anemic. The commonest electrocardiographic abnormalities were (1) changes characteristic of right ventricular hypertrophy in 29 patients and (2) evidence of complete right bundle branch block in 12 patients and incomplete bundle branch block in 7 patients. On roentgenographic examination the following significant abnormalities were noted: right auricular enlargement in 16 cases; right ventricular enlargement in 21 cases; pulmonary artery enlargement in 27 cases, and active pulmonary artery enlargement in 19 cases.

Cardiac catheterization was performed in all cases but two. In the latter the diagnosis was established during operation for other suspected conditions. Catheterization data are not available for review in four of these cases.

The catheter passed through the septal defect in eight patients. All of the patients catheterized exhibited an oxygen saturation difference between the blood of the cavae and the blood of the right atrium. This difference was between 10% and 20% in 25 cases, and it exceeded 20% in three cases.

The pulmonary artery pressure was normal (0–40 mm. Hg) in 9 cases, between 40 and 60 mm. Hg in 15 cases, and exceeded

TABLE 3.—*Heart Characteristics in Thirty-Four Cases in Which Closure of Interatrial Septal Defect Was Attempted*

Size		Murmurs		Rhythm		P_2 Sound		Lungs	
Normal	7	Typical		Normal	31	Split	19	Normal	32
Slightly enlarged	9	Loud	25	Auricular fibrillation	1	Increased	20	Rales	2
Enlarged	18	Soft	8	Extrasystole	1				
		Diastolic	8	Nodal tachycardia	1				
		Atypical	1						

TABLE 4.—*Catheterization in Patients with Interatrial Septal Defect*

Catheterization	Cases, No.
Performed	32
Not done	2
Data not available	4
Of defect	8
Oxygen saturation increased from vena cava to right auricle	
10-20%	25
More than 20%	3

TABLE 5.—*Pulmonary Artery and Right Ventricle Pressures*

Pulmonary Artery Pressure	Cases, No.
0-40 mm. Hg	9
40-60 mm. Hg	15
More than 60 mm. Hg	2
Right Ventricle Pressure	
Normal	1
More than 60 mm. Hg	1
Significant Pressure Gradient	
Right ventricle—pulmonary artery	8

TABLE 6.—*Temperatures of Patients at Operation*

Type of Operation	Temperature	
	Normothermic	Hypothermic
Atrial well	4	10
Atrioseptopexy	12	4
"Open"	0	4

60 mm. Hg in 2 cases. The pulmonary artery was not catheterized in two additional patients; in one the pressure in the right ventricle was normal, and in the other the pressure exceeded 60 mm. Hg.

Of the 17 patients with pressure in the pulmonary artery of more than 40 mm. Hg, 8 had a significant pressure gradient between the right ventricle and pulmonary artery. In six of these eight patients the differential was considered indicative of a relative pulmonic stenosis. The other two patients were thought to have moderate organic pulmonic stenosis.

Anesthesia.—Cyclopropane and ether were the most commonly employed anesthetic gases, although a variety of combinations of anesthetic agents have been used in this series.

Hypothermia produced by external cooling was utilized in 18 operations. In 10 patients the temperature was reduced less than 7 C; 8 patients were cooled below 30 C.

Table 7 indicates the temperatures and periods of circulatory interruption which have been used in this series.

Operative Findings.—The defects in this series have been classified as ostium secundum (high, central, or posterior-inferior), and ostium primum. There were 3 high defects, 17 central defects, 13 posterior-inferior defects, and 1 ostium primum defect.

An incomplete rim was found on the posterior-inferior margin in 10 cases and on the anterior rim of the defect in one case. Transposed pulmonary veins were found in one case with a high defect and in one case with a posterior-inferior type of defect. A persistent left superior vena cava was found in combination with one centrally located defect and with one posterior-inferior defect. In the case of one centrally located defect, the posterior-inferior margin of the defect was fenestrated (Table 8).

Operations.—There were 36 operations for closure of interatrial septal defect performed on 34 patients in this series. There were two deaths which occurred at the time of operation. One death occurred in a 35-year-old male patient on whom an attempt had been made in 1951 to close the defect by invagination of the atrial walls by the "button" technique. After this procedure, the patient developed recurrent attacks of subacute bacterial endocarditis, and it was known that the defect was incompletely closed. He was in poor general condition at the time of reexploration two years later and developed fatal ventricular fibrillation as the skin incision was being made.

TABLE 7.—*Temperatures and Periods of Circulatory Interruption Used in This Series*

Temperature, C	Period of Occlusion, Min.
26.5	8.5
27.0	9.5
29.5	7.5
25.5	7.5

TABLE 8.—*Type of Defect*

Type	Cases, No.
Ostium primum	1
Ostium secundum	..
High	3 *
Central	17 † ‡
Posterior-inferior	13 * ‡

* Transposed pulmonary veins.
† Fenestrated defect.
‡ Persistent left superior vena cava.

TABLE 9.—*Methods Employed in This Series*

Type of Operation	Defect Closed	Not Closed	Deaths
Atrial well	11	2*	2
Atrioseptopexy	15	2†	0
"Open"	4	0	0

* One defect subsequently closed by atrioseptopexy method.
† One defect subsequently closed by atrial-well method.

TABLE 10.—*Immediate Postoperative Complications According to Method Employed*

Complication	Method		
	Atrial Well	Atrio-septo-pexy	Open
Arrythmias	3	2	..
Failure	1	0	1
Pneumonia	1	1	2
Pericarditis	3	0	..
Heart block	2	2	2
Transient Mental confusion	..	..	1
None	7	10	..

The second death occurred in the case of a very ill six-year-old girl. Approximately 3 minutes after the atrial well was opened, a three-to-one heart block developed and this was followed by intractable cardiac arrest. There was no satisfactory explanation of this series of events. Overfilling of the heart with blood from the "well" was considered as a possibility. The last 29 patients were operated upon without mortality.

An attempt was made in the patient with the ostium primum defect to close the opening with a nylon patch. During the ensuing 16 days, the child developed progressive intractable failure. The patch was removed on the 16th postoperative day, and the patient gradually returned to her original preoperative condition.

An attempt was made to close the defect by the Gross atrial "well" technique in 15 patients. The atrioseptopexy method was used in 17 patients, and the "open" repair was used in four patients (Table 9).

The immediate postoperative complications are tabulated in Table 10 according to the method of closure employed.

Postoperative Evaluation.—There is no information available on the postoperative condition of two patients, and in three other patients the postoperative data are not adequate for evaluation of their present condition. Of the 27 patients available for evaluation, 17 are considered improved, 2 are somewhat improved but continue to complain of dyspnea, 3 were asymptomatic prior to operation and are unchanged, and in 5 cases the postoperative period has been too brief for evaluation (Table 11).

For evaluating the postoperative condition of the patient, the following criteria have been utilized: persistence or reoccurrence of preoperative symptoms, heart size by roentgenogram, electrocardiogram, and status of murmur.

The postoperative period of all patients operated upon by the "open" method has been too short for evaluation of the final result. One child, six weeks after operation, continues to have a loud systolic murmur and thrill to the left of the sternum. The early progress of the other three patients has been satisfactory.

Of the 20 patients improved or unchanged who at present are asymptomatic, 9 had repair of the defect by the atrial-well method and 11 by the atrioseptopexy technique. Two patients in whom the defect was not closed are considered unimproved. In one of these patients an attempt was made to close the defect by the atrial-well technique, and in the other closure was attempted by the atrioseptopexy method.

COMMENT

The indications for closure of an interatrial septal defect are at present ill defined. The symptoms in the majority of the cases in this

TABLE 11.—*Postoperative Evaluation of Patients*

Postoperative Condition	Patients, No.
Operative deaths	2
Not followed	2
Inadequate data	3
Too soon for evaluation	5
Improved	17
Unchanged (asymptomatic)	3
Unimproved (defect not closed)	2
Total	34

series were not severe, although we are inclined to agree with Bailey [3] that careful questioning in many supposedly asymptomatic patients will reveal definite symptoms, limitations, and self-imposed restrictions of activities. The diagnosis in all cases in the present series was established by cardiac catheterization or surgical exploration.

Criteria which have been considered important as indications for surgical intervention have included the following:

1. Disabling or progressive symptoms, usually related to fatigability or dyspnea.

2. Evidence of pulmonary hypertension or a left to right intracardiac shunt of considerable magnitude.

3. Roentgenographic evidence of cardiac enlargement and signs of right ventricular strain by electrocardiogram.

4. A history of recurrent episodes of upper respiratory tract infections or pneumonia. All of our patients have had at least two of these findings.

It is known that medial hypertrophy and intimal sclerosis of the pulmonary arterioles and pulmonary hypertension may occur with long-continued increased pulmonary blood flow. These changes may become progressive and irreversible, leading to a serious embarrassment of pulmonary function. In patent interauricular septal defects this situation is prone to occur in the third and fourth decade. With this consideration in mind, operation has been recommended in certain relatively asymptomatic patients.

In this series satisfactory closure of these defects has been accomplished by three methods. Atrioseptopexy seems the easiest and least hazardous procedure. This technique seems best adapted for closure of the small or moderate-sized centrally located defect. The atrial-well technique is more cumbersome and has the disadvantage of also being a "blind" technique. Our experience with the open method has been limited. The procedure requires a bilateral thoracotomy and complete interruption of circulation; hence it is an operation of great magnitude. We agree with Swan and Lewis, however, that by means of this technique the operator may be more certain that a secure closure has been obtained and that certain complicated defects are best visualized and closed.

At present our procedure is to externally cool the patient to a temperature below 30 C. The chest is prepared for a bilateral thoracotomy. The right thorax is opened through the fourth interspace anteriorly, and the septum is explored by the index finger introduced through the right atrial appendage. If the defect lends itself to closure by the atrioseptopexy method, this type of closure is performed; if not, the incision in the appendage is closed and the thoracotomy incision is extended into the left thorax. When the patient's temperature is reduced to between 25 C and 28 C, the circulation is interrupted, the right auricle is opened widely, and the defect is closed with a continuous nonabsorbable surgical (silk) suture.

Drugs or chemicals have not been used to alter cardiac action during or after the period of inflow occlusion. Instead, we have relied entirely upon the early reestablishment of an adequate coronary blood flow, by either normal cardiac contractions or cardiac massage when necessary. If ventricular fibrillation occurs during the occlusion period, no attempt is made to correct it. Immediately upon release of the occluding tapes, however, cardiac massage is begun. This is continued until an adequate coronary blood flow is assured and the tone of the cardiac muscle returns. Defibrillation is then accomplished by two or three electric shocks. This is followed by a sinus rhythm.

SUMMARY

A review is presented of the diagnosis and treatment of 34 patients with interatrial septal defects.

Thirty-six operations were performed for closure of the defects in these 34 patients. There were two deaths during operation. The last 29 patients have been operated upon without a mortality.

The defect was successfully closed in 30 patients. Eleven defects were closed by the atrial-well technique, fifteen by the atrioseptopexy method, and four by the "open" technique utilizing cardiac inflow and out-

flow, occlusion, and hypothermia. The atrioseptopexy method is currently employed when possible to close the uncomplicated centrally placed defects. The "open" technique is utilized for the closure of defects that cannot be satisfactorily repaired by atrioseptopexy.

The postoperative result is considered clinically satisfactory in 20 patients. Data are not available for proper evaluation of the result in five cases. Five other patients have been operated upon too recently for evaluation.

REFERENCES

1. Muller, W. H., Jr.; Smith, S. W.; Dammann, J. F., Jr.; Adams, F. H., and Darsie, M. L.: Considerations and Physiologic Studies in the Closure of Interauricular Septal Defects, Surgery **37**:1, 1955.

2. Taussig, H. B.: Congenital Malformations of the Heart, New York, The Commonwealth Fund, 1947.

3. Bailey, C. P.; Downing, D. F.; Geckeler, G. D.; Likoff, W.; Goldberg, H.; Scott, J. F.; Janton, O., and Redondo-Ramirez, H. P.: Congenital Interatrial Communications: Clinical and Surgical Considerations with a Description of a New Surgical Technic: Atrio-Septo-Pexy, Ann. Int. Med. **37**:888, 1952.

4. Lewis, F. J.; Taufic, M.; Varco, R. L., and Niazi, S.: The Surgical Anatomy of Atrial Septal Defects: Experience with Repair Under Direct Vision, Ann. Surg. **142**:401, 1955.

5. Cohn, R.: An Experimental Method for Closure of Interauricular Septal Defects in Dogs, Am. Heart J. **33**:453, 1947.

6. Bailey, C. P.; Bolton, H. E.; Jamison, W. L., and Neptune, W. B.: Atrio-Septo-Pexy for Interatrial Septal Defects, J. Thoracic Surg. **26**:184, 1953.

7. Cooley, D. A.: Surgical Closure of Atrial Septal Defects, Surg. Gynec. & Obst. **100**:268, 1955.

8. Gross, R. E.; Watkins, E., Jr.; Pomeranz, A. A., and Goldsmith, E. I.: A Method for Surgical Closure of Interauricular Septal Defects, Surg. Gynec. & Obst. **96**:1, 1953.

9. Blount, S. G., Jr.; McCord, M. C., and Swan, H.: Surgical Closure of Atrial Septal Defect: Response in a Patient with Severe Pulmonary Hypertension, Am. Surgeon **20**:305, 1954.

10. Blount, G., Jr.; Swan, H.; Gensini, G., and McCord, M. C.: Atrial Septal Defect: Clinical and Physiologic Response to Complete Closure in 5 Patients, Circulation **9**:801, 1954.

11. Lewis, F. J., and Taufic, M.: Closure of Atrial Septal Defects with the Aid of Hypothermia: Experimental Accomplishments and Report of One Successful Case, Surgery **33**:52, 1953.

12. Lewis, F. J.: Repair of Atrial Septal Defects During Hypothermia, Postgrad. Med. **17**:293, 1955.

DISCUSSION

On Papers by Drs. Davis and Fell; Gerbode, and Longmire and Smith

Dr. John Michael Dorsey, Evanston, Ill.: I cannot resist the temptation to say that these papers are of inestimable value as far as this Association is concerned because of their clarity. Even those of us who may or may not do cardiac surgery cannot help being benefited by the clear presentations of, first, the defects which mimic patent ductus, as Dr. Davis has shown us; second, the various unusual types of ductus that Dr. Gerbode has pointed out to us, and, third, the operation Dr. Longmire has shown.

I certainly wish to compliment these gentlemen on informing us about these three very important subjects.

Dr. Oscar T. Clagett, Rochester, Minn.: I really cannot add too much to the three presentations. I believe our experience is very similar to theirs. With increasing experience with a fairly large number of patent ductus cases, it is inevitable that we do encounter these unusual varieties and conditions that simulate patent ductus, as Dr. Davis mentioned.

We have had similar experiences, too, in that in some of these cases we failed to establish a completely accurate preoperative diagnosis. With better cardiac catheterization data, mistakes regarding the condition you are dealing with fortunately become rare. It is essential in this type of surgery to know what you are dealing with. Certainly surgeons have much to be grateful for in having the cardiologists and techniques of diagnosis that are as accurate as they are now.

I was interested in Dr. Gerbode's paper because we have also been interested in pulmonary hypertension, and I believe our experience is similar to his. I do not believe we understand fully why some patients with patent ductus have hypertension and others do not. It is an important factor surgically, and it is something that should be recognized preoperatively.

I believe it is one reason for advising an operation in people with patent ductus relatively early and not delaying the operation unnecessarily, because, in my opinion, some of these pulmonary hypertension changes are probably progressive and can become extremely serious.

We have been impressed by another factor that Dr. Gerbode brought out, namely, the association

of a patent ductus and coarctation. For some reason patent ductus tends to occur twice as often in females as in the males, and coarctation occurs three or four times as commonly in males as in females, according to our experience, yet in 16% of our experiences with coarctation there has been an associated patent ductus. It is an important factor in diagnosis and treatment. By and large, these patients with associated anomalies have responded well to surgical treatment.

Certainly Dr. Longmire's presentation was beautiful. The motion picture he showed is one of the finest I have seen. I believe you might be interested in the experience of my associate, Dr. Kirklin, with intracardiac lesions of that kind. He has preferred the well technique that Dr. Longmire mentioned, rather than hypothermia; but now that the mechanical age has arrived, he has at the present time operated on about 40 patients with a heart-lung machine of the Gibbon type, for various intracardiac defects, and it is working out extremely satisfactorily.

Dr. Carl B. Davis Jr., Chicago: I certainly agree with all that Dr. Gerbode has said about patent ductus arteriosus. We also have seen cases of what we like to call malignant patent ductus arteriosus in children under the age of 2 years, who have had much the same findings as have been presented. Most certainly these children should be operated upon before they succumb from heart failure.

I should like to reiterate what Dr. Clagett and Dr. Dorsey have said, namely, that Dr. Longmire's motion picture was very well presented. I wish to thank Dr. Dorsey for his laudatory remarks and to say that I appreciate very much the discussion of Dr. Clagett.

Dr. William P. Longmire, Los Angeles: I should like to say that, of course, the problem of pulmonary hypertension, which Dr. Gerbode brought up, is also one of considerable importance in a case of interauricular septal defect. It is one reason why we believe at the present time that one should consider closing these defects in the relatively asymptomatic patient.

I also should like to thank the discussers. Dr. Clagett mentioned the work of Dr. Kirklin. It is certainly true that Dr. Kirklin has been one of the first to demonstrate the practicality of utilizing artificial means of cardiac action and oxygenation for cardiac surgery. To my mind there is no question but that this is the future trend in cardiac surgery.

Molded, Seamless Tubes of Polyvinyl Formalinized Sponge

Their Use as Replacements for Segments of Aorta and Major Arteries

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and
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Currently among surgeons and investigators interested in vascular surgery there is a widespread interest in attempts to produce an entirely satisfactory synthetic vascular prosthesis. Most work [1] has been done with cloth tubes made from synthetic fibers such as Vinyon-N, nylon, Dacron, Orlon, and so forth. Although results have been generally fairly good and are improving as better products are being developed and are being used more effectively, it seems generally agreed that vascular grafts made from these cloth fabrics are not ideal and can probably be improved on.

For several years one of us (Grindlay) has been investigating tissue responses to various forms of polyvinyl formalinized (Ivalon *) sponge [2] implanted into many sites in experimental animals and in certain sites in clinical patients. As early as 1950, he fashioned blood-vessel grafts of this material and in pilot studies implanted them in the aortas of dogs. During the past year, after a simpler and more effective method of forming the sponge into artificial arteries had been devised, we have carried out a more formal investigation of Ivalon vascular prostheses. Shumway and associates [3] have recently reported favorable experience with similar compressed Ivalon vascular prostheses.

Read at the 63d Annual Meeting of the Western Surgical Association, Seattle, Dec. 2, 1955.

Fellow in Surgery, Mayo Foundation (Dr. Mortensen), and Section of Surgical Research, Mayo Clinic and Mayo Foundation (Dr. Grindlay). The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

* Polyvinyl formalinized sponge (surgical Ivalon sponge) used in this study was furnished by the manufacturer, Ivano, Inc., Chicago, and is obtainable from Clay Adams, Inc., New York.

METHOD OF CONSTRUCTION OF IVALON VASCULAR PROSTHESIS

The first step in making a compressed Ivalon vessel is the construction of a model of the lumen of the vessel with the desired size, shape, and branches. Glass or metals which have a smooth hard surface are most satisfactory. If branches are desired, as in the aortic bifurcation, the model must be so constructed that the branches are detachable (Fig. 1) so that they can be removed and the intact graft slipped off the model. The external surface of the model must be smooth, and all joints must be well tooled without cracks or defects, since the inner surface of the completed graft will be an exact cast of the external surface of the model.

After being thoroughly rinsed, the dried or frozen brick of Ivalon sponge is prepared for use by slicing off sheets 2 mm. thick, which are then cut into strips 3 to 5 cm. wide (Fig. 2*a*). A special double-bladed borer powered by a drill press is used to produce seamless tubes of Ivalon sponge (Fig. 2*b*). These tubes and thin strips of sponge are the raw materials used to make the grafts. An Ivalon sponge tube of the appropriate size is pulled over the model. When a graft for the lower aorta and bifurcation is to be made, several tubes are required for the main vessel and for each branch; before wrapping is begun, these tubes are joined together by interrupted nonabsorbable surgical (cotton) sutures. Then the strips of Ivalon are wrapped around the tube-covered model, much as a roller bandage or plaster cast is applied (Fig. 3). The Ivalon is applied while stretched under considerable tension to provide compression. Usually three layers of 2 mm.-thick Ivalon sponge strips on top of the inner tube of sponge, compressed about fourfold to sixfold to a final thickness of 1.5 to 2 mm., give the desired

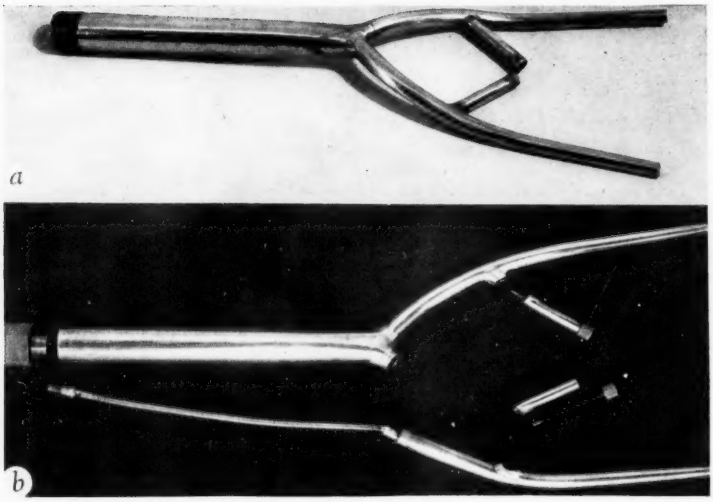
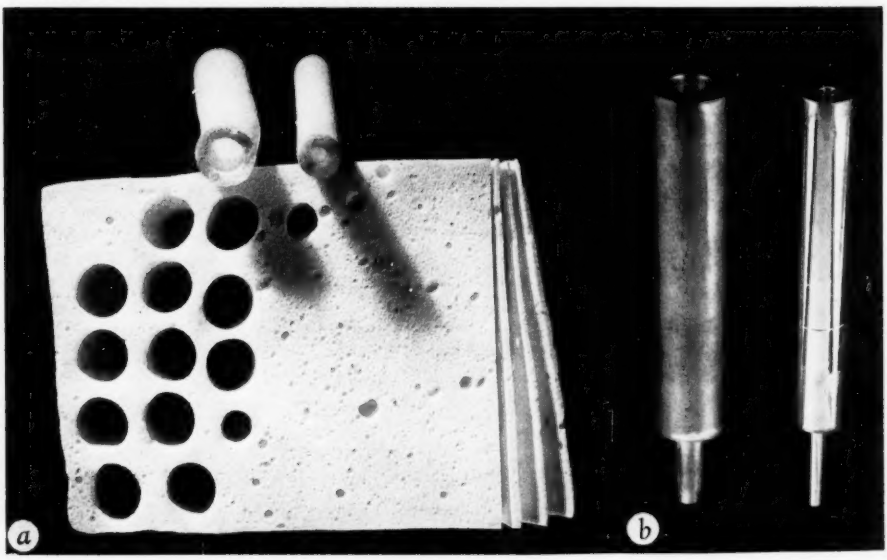


Fig. 1.—Brass model used for construction of prosthesis of aortic bifurcation. *a*, The model assembled, ready for wrapping. *b*, The model disassembled, allowing it to be slipped out of the polyvinyl-formalinized-sponge prosthesis.

properties to the finished prosthesis. When one strip of Ivalon has been wrapped on the model, another is started, the ends being overlapped slightly but not otherwise joined together. At points where wrapping is irregular, such as at bends or at the origins of branches, extra wrapping is advisable to assure at least four layers of Ivalon. When the model has been adequately and properly covered with Ivalon sponge strips, additional compression, control of loose ends, and prevention of unwrapping are achieved by a final snug wrapping with a 2-in. gauze bandage.

Fig. 2.— *a*, Preparation of sheets, strips, and tubes of polyvinyl formalinized sponge from the block in which it is supplied. *b*, Special double-bladed borer used to produce tubes of polyvinyl formalinized sponge.



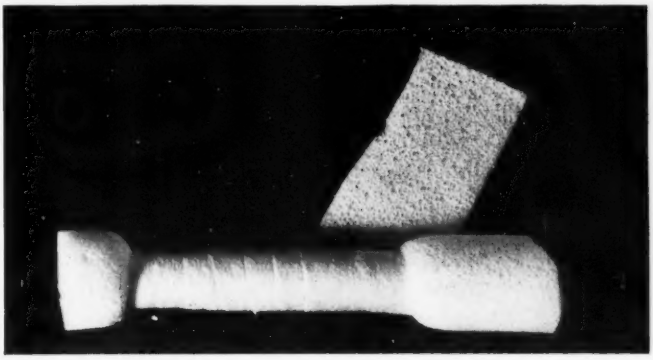


Fig. 3.—Wrapping strips of polyvinyl formalinized sponge around a glass model, which is first covered by a seamless polyvinyl-formalinized-sponge tube.

The entire preparation, consisting of the model covered first by a seamless tube of Ivalon, then by wrapping of Ivalon strips, and finally by an outer layer of gauze bandage wrapping, is then boiled in distilled water for 20 minutes or autoclaved moist (submerged in water) at 250 F for 20 minutes. This heat treatment serves to sterilize the prosthesis, fuses the layers of Ivalon sponge into a seamless one-piece prosthesis, and "sets" the sponge to the size, shape, contour, and degree of compression at which it was maintained while heated. After the boiling or autoclaving, the prosthesis on its model is allowed to cool under aseptic conditions. The gauze wrapping is then removed aseptically, and the sterile prosthesis is slipped off its model after any branches of the model have been disconnected and separately removed. The graft is then ready for implantation. It can be stored indefinitely if kept sterile. It will slowly dry, becoming stiff and hard, but can be reconstituted by simply soaking for a few minutes in sterile water or saline solution. The prosthesis must not be reboiled or autoclaved again or it will return to its original state, consisting of uncompressed, unfused strips of Ivalon sponge.

PROPERTIES OF COMPRESSED POLYVINYL-FORMALINIZED-SPONGE VASCULAR PROSTHESES

Blood-vessel grafts made as described above possess certain unique and desirable physical properties not found in other types of vascular prostheses. The finished Ivalon vessel closely resembles, physically, a fresh normal aorta; in several aspects it is superior, so far as surgical implantation is concerned.

1. *Elasticity and Pliability.*—Perhaps the most significant and desirable property of a compressed Ivalon prosthesis is its elasticity. The prosthesis stretches in all directions to a similar degree and with approximately the same resistance as does a normal vessel or aorta (Fig. 4). This feature allows the Ivalon graft to pulsate when carrying blood during the initial phase of implantation, permits easy fits at anastomoses without formation of creases or wrinkles, and makes it possible to bend the vessel without compromising its lumen or producing kinks or wrinkles. These, of course, have been chief disadvantages of the cloth-tube prostheses.

2. *Porosity and Permeability.*—A properly made Ivalon vascular prosthesis possesses the ideal degree of porosity. It will hold water not under pressure but is permeable to water at about 20 mm. of pressure. When blood is first allowed to fill the implanted Ivalon graft under arterial pressure, there will occasionally be a slight oozing of serum or blood through the wall for a few seconds but never to an extent comparable to the bleeding regularly seen through the walls of a freshly implanted cloth prosthesis. Interstices between meshes of the compressed Ivalon prosthesis are, however, sufficiently patent to permit ready ingrowth of vascularized fibrous tissue. In addition, this ingrowth of tissue is aided materially by the hydrophilic properties of the Ivalon itself.

3. *Strength.*—Although studies of tensile strength have not been done, the Ivalon pros-

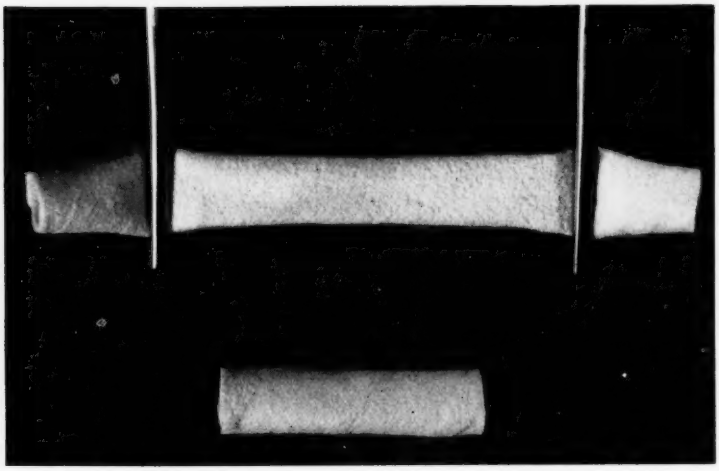


Fig. 4.—Elasticity of polyvinyl-formalinized-sponge vascular prosthesis. The prosthesis has been stretched to almost double its normal length.

thesis is considerably stronger than the normal aorta. This is particularly apparent during suturing. Tiny sutures only 1 to 2 mm. from the edge of the Ivalon prosthesis do not pull out, the arterial nonabsorbable surgical (silk) suture breaking before the Ivalon tears through. It is important to realize that as time passes after implantation of the prosthesis it does not become weaker, since it is not absorbed, as homografts are; on the contrary, it becomes stronger as tissue invades its interstices.

4. *Seamlessness.*—Because the innermost layer is a seamless tube and because the sterilization process fuses the inner tube and outer layers of Ivalon into a single continuous mass, there are no seams or weak regions in the prosthesis prepared as described above. This obviates two important disadvantages of the seamed cloth tubes, namely, tendency to rupture as seams fray and weaken and thrombosis at the roughened seams.

5. *Smooth Inner Surface.* — After the sterilization process, the Ivalon which has been held snugly against the smooth hard surface of the model develops a smooth inner surface resembling the shiny interior of a normal artery. This smooth inner surface decreases the likelihood of thrombosis within the graft.

6. *Resiliency.*—Molding of Ivalon over the model causes the prosthesis to have a tubular, rounded contour in cross section. Thus, the ends of the prosthesis stay open while the anastomosis is being made, instead of collapsing, as do the cloth prostheses as well as homografts. This property of Ivalon prostheses makes accurate anastomosis easier, tends to prevent constrictions at suture lines, and makes it difficult or impossible to suture the vessel closed, which, unfortunately, is possible when one is using less resilient grafts.

7. *Thickness of Wall.*—An Ivalon prosthesis constructed as described above has a wall only slightly thicker than that of normal aorta. This makes use of cuffs, rolled edges, or folded-over anastomoses unnecessary. Simple running coaptation sutures are entirely adequate and satisfactory in making anastomoses to Ivalon prostheses.

One word of caution must be sounded concerning Ivalon vascular prostheses: They must be properly constructed. All the properties described above are relative and variable, depending on proper attention to details in preparation of the prosthesis. For example,

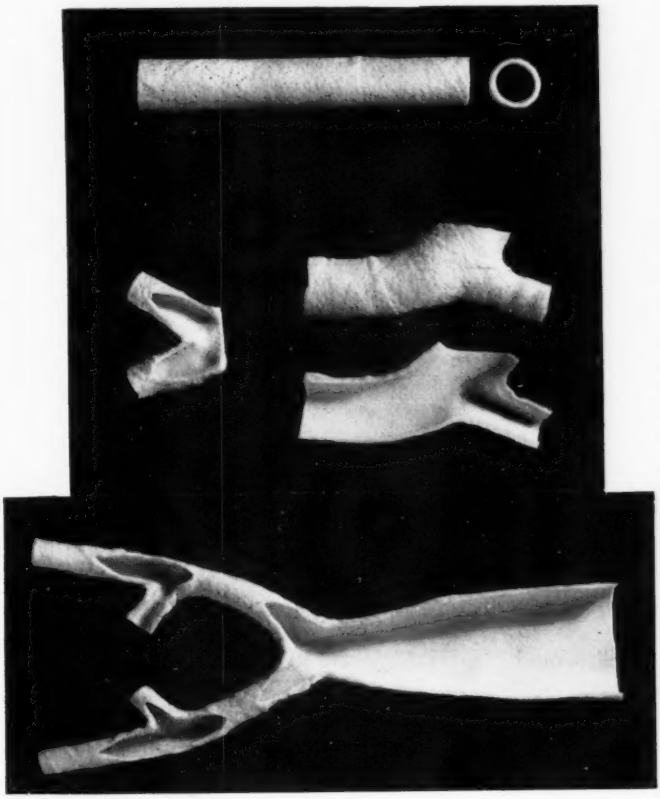


Fig. 5.—Types of polyvinyl-formalinized-sponge vascular prostheses which have been surgically implanted. *Top*, straight (slightly tapered) tubes for abdominal aorta, descending thoracic aorta, and peripheral arteries. *Left, middle*, aortic bifurcation. *Right, middle*, distal aortic arch, including left subclavian branch. *Bottom*, aortic bifurcation (human), including bifurcation of each common iliac artery (opened, to show smooth lining).

if the graft is made with walls that are too thick or too severely compressed, the elasticity, flexibility, and permeability to tissue ingrowth will be sacrificed. On the other hand, if the Ivalon is not wrapped evenly, if the walls are too thin, or if insufficient compression is maintained during the heating process, the graft will not have adequate strength or resiliency, will be too porous and have a tendency to leak, or may dilate or even rupture under arterial pressure.

Biologically, Ivalon is well tolerated by human tissues. There is no excessive fibrosis and no foreign-body reaction if the Ivalon is clean and free from foreign material. Its hydrophilic and nonirritative properties permit it to become thoroughly invaded with vascularized fibrous tissue, to act, in other words, as a framework for "living tissue."

SURGICAL RESULTS

Vascular prostheses of compressed Ivalon (Fig. 5) have been implanted in the abdominal aorta, thoracic aorta, aortic trifurcation, including iliac vessels no more than 3 mm. in diameter, aortic bifurcation in which the common internal iliac vessel was ligated and the bifurcation graft sutured to both external iliac vessels, and aortic arch, including the left subclavian branch in a total of 17 dogs (Table). Animals have been killed and grafts examined grossly and histologically at intervals up to 11 months after implantation. Aortograms have been made on all animals not killed in order to demonstrate the condition of the graft. To date, none of these compressed, seamless Ivalon vascular prostheses has thrombosed, none has leaked, ruptured, or dilated postoperatively, and none

Site of Implantation of Seventeen Seamless, Compressed Polyvinyl-Formalinized-Sponge Vascular Prostheses

Site	Grafts, No.
Distal aortic arch, including left subclavian artery	4
Descending thoracic aorta	4
Abdominal aorta	4
Terminal aorta, bifurcation, and external iliac artery	3
Terminal aorta and trifurcation	2
Total	17

has been poorly accepted or tolerated by the recipient.

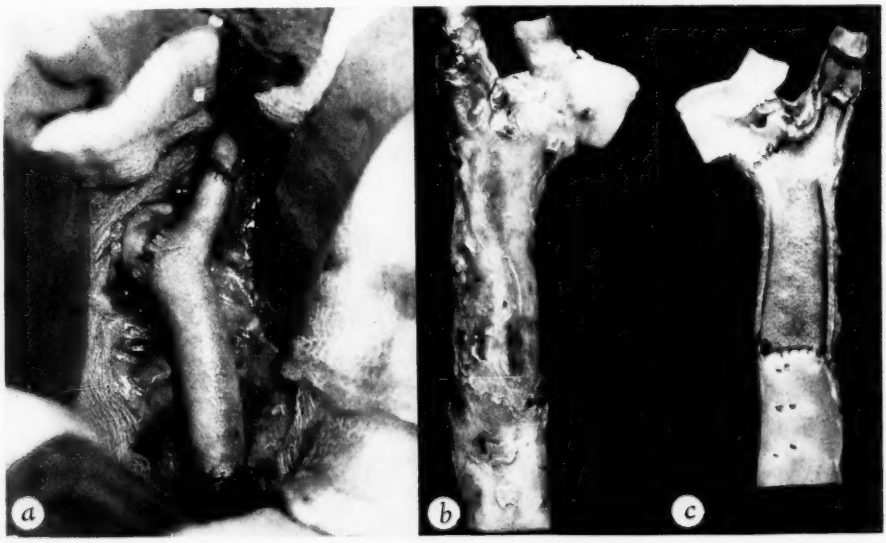
Because of the excellent results in experimental animals and the extensive favorable experience with Ivalon implanted in other human organs and tissue, these seamless compressed vascular prostheses have been used in approximately 15 clinical patients requiring resection of segments of the aorta and its major branches. To date, we are aware of no unfavorable sequelae related to the Ivalon grafts used clinically.

HISTOLOGIC SEQUELAE

Within a few hours after implantation, fibrin and trapped cellular elements of blood are demonstrable in the interstices and on the outer surface of the Ivalon prosthesis. Fibrocytes, capillary buds, and collagen fibers are present within two weeks. These tissues are most abundant on the external surface of the prosthesis and invade the interstices of the wall from outside inward. Within three to five weeks vascularized fibrous tissue is found all the way through the wall to its inner surface (Fig. 6). This fibrous tissue gradually thickens, and its collagen content increases, reaching a stable state of pliable, tough vascularized fibrous impregnation of the wall of the prosthesis in four to six months.

The inner surface of the prosthesis, being smooth and glazed, collects its fibrin coating or newly formed inner lining at a rate quite similar to that noted in homografts. This is considerably slower than the rate at which fibrin and fibrocytes form on the comparatively rough, woven surface of cloth prostheses, which have been shown to collect a fibrin layer within a few hours after implantation. A thin internal layer of fibrin, mesenchymal cells, and fibrocytes is usually found lining an Ivalon prosthesis about two weeks after implantation. This layer of cells thick-

Fig. 6.—Appearance of polyvinyl-formalinized-sponge vascular prostheses which have been implanted surgically. *a*, Distal aortic arch and subclavian artery carrying blood under arterial pressure, immediately after implantation. *b* and *c*, Prosthesis seen in *a*, after being in place for six weeks. *b*, External appearance. *c*, Internal appearance.



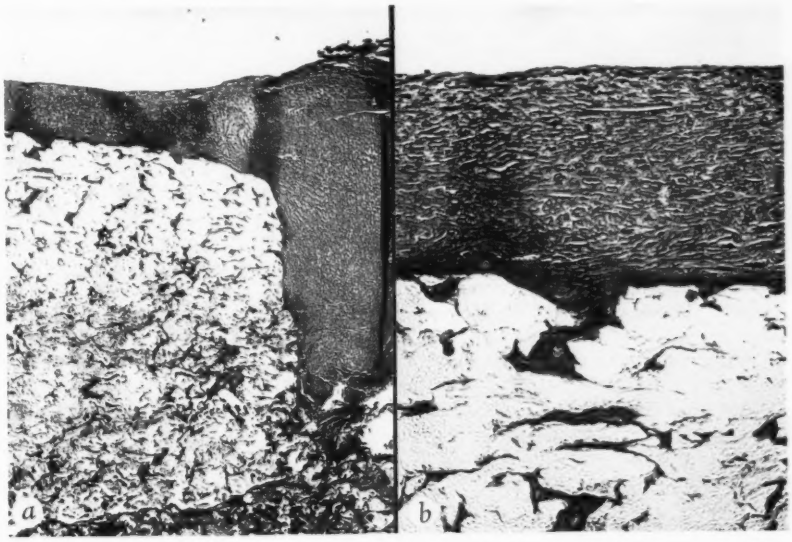


Fig. 7.—Histologic appearance. *a*, Anastomosis of polyvinyl-formalinized-sponge prosthesis six weeks after implantation (hematoxylin and eosin; reduced slightly from mag. × 80). Note well-developed, newly formed "intima" over the compressed polyvinyl-formalinized-sponge prosthesis and the fibrous tissue invading the interstices of the prosthesis. *b*, Portion of wall of a polyvinyl-formalinized-sponge prosthesis implanted for eight weeks. The lumen is at the top (hematoxylin and eosin; reduced slightly from mag. × 180).

ens for two or three weeks until it reaches a depth of 8 to 10 cells (Fig. 7*a*). Then the individual cells become modified into flatter, more closely packed units resembling actual intima. After six months this newly formed internal layer of cells has become stable and is seen to be continuous with the normal interior of the artery proximal and distal to the prosthesis (Fig. 7*b*).

SUMMARY AND CONCLUSIONS

Seamless tubular prostheses made of compressed Ivalon sponge can be formed in any desired size and shape, with or without branches. These vascular prostheses have physical and biologic properties that closely resemble arterial homografts, overcoming most of the disadvantages of the better-known prostheses made of synthetic fiber cloths. Such compressed Ivalon prostheses have been well tolerated and remarkably free from complications and disadvantages when implanted as aortic and arterial grafts in experimental animals. A limited number of these vascular prostheses have been implanted as aortic and arterial grafts in human patients and appear to have given satisfactory results.

DISCUSSION

Dr. James Barrett Brown, St. Louis. I should like to mention a few things about polyvinyl formalinized sponge that might back up the work of Drs. Mortensen and Grindlay and make us feel freer about using it in relation to possible tumor formation.

We have observed more than 100 animals through their entire life cycle and have found no tumor formation following the implantation of this polyvinyl formalinized sponge. We believe it is free of that tendency. On the other hand, polyvinyl chloride does seem to give some trouble.

The Oppenheimers have shown that even polyethylene causes some tumor formation, but they did not show that this polyvinyl-alcohol caused it, as far as I know at this time.

There have been other troubles with the sponge when used in large-size bulk.

An interesting thing also about this sponge is that presumably it has a form memory of its shape at about 95 F, and it would seem that the prosthesis keeps the definite form as used by the essayists as determined by its form as it cooled below 95 F.

This form-memory process is the same thing that was seen in airplanes during the early days of World

War II, when it was found that some of the plane fronts would straighten out. They had been fabricated into curved positions but retained through their form memory the original flat plate idea, and the sheets tended to flatten out again and to pop out of place.

This interesting physical finding that this material retains its form memory of its form at around 95 F is another thing to back up the essayists' statements that these materials do retain a good form and stay there.

The term "Ivalon" is a trade mark or patent to signify polyvinyl formalinized sponge. I know of only one company that makes it, and it has been well accepted.

I should like to congratulate the essayist and Dr. Grindlay for the work they have done.

DR. HAROLD LAUFMAN, Chicago: In the smaller diameters—let us say that of the popliteal artery—does this material retain its elasticity after a year, and could it be used in the area of a joint? Most of the other materials become rather brittle after 10 to 15 months and become occluded when they are used across a joint, especially in the smaller diameters.

DR. JOHN H. GRINDLAY, Rochester, Minn.: In answer to Dr. Brown's question whether the material has elastic memory if reheated to a point above 95 F, I am sure his question can be answered in the affirmative. The polyvinyl formalinized sponge will return to the shape and position of the material from which it was made. The tubes would not maintain the shape you saw in the slides when the material was formed by moist heat.

In answer to Dr. Laufman's question, the material does not have elasticity after it has been invaded by fibrous tissue. It has the same type of elasticity that a mass of fibrous tissue of the same size and shape would have but not the elasticity of the aorta. Perhaps that is a disadvantage.

As I see it, the advantage of this material over other vascular grafts—for instance, the advantage over a homograft—is that there is no material that has to be phagocytized and removed. The implant is ready for invasion at the moment it is implanted—and invasion begins very quickly. With the homograft, as you know, the tissue must be removed and replaced by the host tissue.

I do not believe any material has yet been tested sufficiently long to allow us to say it is the perfect replacement for a blood vessel. This includes homografts and autografts, and it certainly is the case with any of the newer synthetic materials. We have not observed them long enough.

We hope that perhaps a better material will be found that will be the perfect one, if this is not.

DR. JD MORTENSEN, Rochester, Minn.: I appreciate the comments very much. I believe Dr. Grindlay has answered accurately the questions that were raised.

I might add that it is quite true that the plastic material has elastic memory for its previous condition, before it was treated with heat, but at body temperatures over a prolonged period this factor does not come into play. It requires considerably more heat than is present in the body to permit the Ivalon to return to its original state.

As far as the elasticity of these grafts is concerned, after they have been implanted for some time they do become invaded with fibrous tissue, which, as you know, is inelastic. Elastic fibers have never been seen in this type of prosthesis, nor any other in homografts, for that matter. However, they are as flexible as or more flexible than homografts and have been used in extremities, including the popliteal region, as suggested. We have seen no evidence that the diameter of the vessel has any effect on its ability to become stiff or maintain pliability. While there may be inelasticity, there is a certain degree of pliability which remains as the prosthesis contains and becomes fibrous tissue.

In closing, I should like to pay special tribute to Dr. Grindlay for his guidance during the preliminary work that was carried on, for his keen interest and valuable suggestions throughout the course of this study, and for the very valuable contribution he has made in experimental surgery in general, particularly in recent years with the use of these synthetic materials.

REFERENCES

1. Deterling, R. A., Jr., and Bhonslay, S. B.: An Evaluation of Synthetic Materials and Fabrics Suitable for Blood Vessel Replacement, Surgery **38**:71-91 (July) 1955.

2. Grindlay, J. H., and Waugh, J. M.: Plastic Sponge Which Acts as a Framework for Living Tissue: Experimental Studies and Preliminary Report of Use to Reinforce Abdominal Aneurysms, A. M. A. Arch. Surg. **63**:288-297 (Sept.) 1951.

3. Shumway, N. E.; Gliedman, M. L., and Lewis, F. J.: An Experimental Study of the Use of Polyvinal Sponge for Aortic Grafts, Surg. Gynec. & Obst. **100**:703-706 (June) 1955.

Portal Hypertension in Infants and Children

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THOMAS B. PATTON, M.D.
and
CLIFFORD D. BENSON, M.D., Detroit

The surgical care of infants and children with portal hypertension has been reported infrequently. It was our desire to add our experience to that already reported and to consolidate what appears to us to be the basic problems facing the surgeon in the diagnosis and care of these children.

PATHOGENESIS

The classification of Whipple [22] with slight modification serves equally well in children as in adults.

I. Prehepatic
   A. Obstruction of inferior vena cava—congenital

II. Hepatic
   A. Portal cirrhosis
   B. Thrombosis of hepatic veins (Chiari's syndrome)

III. Posthepatic
   A. Congenital
      1. Cavernomatous transformation of portal vein
      2. Congenital stricture and obliteration of portal vein
   B. Acquired thrombosis of portal vein or its tributaries
      1. Trauma
      2. Infection
      3. Idiopathic or spontaneous origin

The posthepatic cases are the most distressing to the surgeon, and, although from a clinical standpoint they can be differentiated into congenital and acquired, they are too often indistinguishable.

The etiology of congenital atresia of the portal vein probably is related to changes in the circulation which occur at birth. In the fetus the vitelline veins, the left umbilical vein, and the portal vein unite at the point where the ductus venosus (Arantii) originates to shunt placental blood around the liver to the inferior vena cava. Immediately after birth, the umbilical vein collapses and the ductus venosus begins to close. The ligamentum teres and the ligamentum venosum are formed by the process of fibrotic obliteration (Fig. 1). This obliterative process may involve the entire portal vein or only a small segment, resulting in a stricture. To carry this embryologic explanation [6] one step further, one can explain the rare occurrence of stenosis of the inferior vena cava at the entrance of the ductus venosus on this basis. This fibrotic process extending unchecked into the inferior vena cava via the ductus venosus results in a stenosis or coarctation.

We have seen one (Fig. 2) such case which we believe is a true congenital coarctation of the inferior vena cava, and we find some support for this assumption in the other five reported cases.*

The differentiation between cavernomatous transformation and portal thrombosis (acquired or congenital) into different and distinct entities is at times difficult. Klemperer [7] thought cavernomatous transformation was due occasionally to an actual angioma. However, congenitally the portal vein may not develop as a single large vessel but is made up of many small tributaries. The commonest cause is certainly due to the formation of collateral branches following thrombosis of the portal vein.

Childrens Hospital of Michigan.

Read at the 63d Annual Meeting of the Western Surgical Association, Seattle, Dec. 2, 1955.

Veterans Administration Hospital, Seattle, Washington, and University of Washington School of Medicine (Dr. Jordan); Medical College of Alabama (Dr. Patton).

* References 12-16.

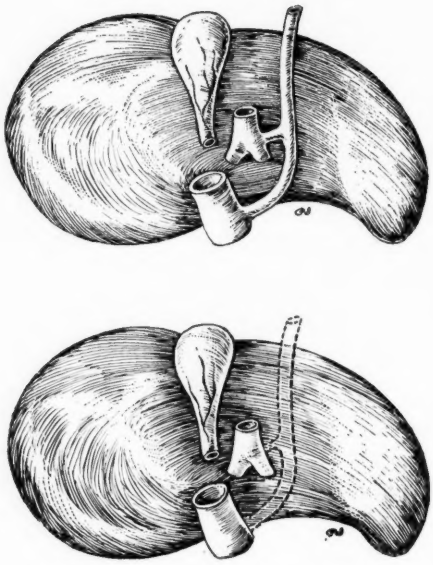

Fig. 1.—Embryology of left umbilical vein and ductus venosus.

TABLE 1.—*Klemperer Cases (Twenty-Three)*

Decade, Yr.	No. Cases	Decade, Yr.	No. Cases
10-20	1	40-50	7
20-30	4	50-60	5
30-40	5	60-70	1

If the etiology is of a congenital nature, the bleeding may occur at an earlier age than if on an acquired basis, and Klemperer's cases of cavernomatous transformation give some credence to this line of thought.

Cirrhosis of the liver is not a rare lesion in children or even infants. Discarding cirrhosis secondary to a biliary obstruction, Gross [4] found the etiology most frequently to be obscure, and the first symptoms were often those of portal hypertension. He states that any process either congenital or acquired which causes liver cirrhosis is capable of producing "intrahepatic block." Hepatitis of either an infectious or a toxic nature is the commonest cause.

DIAGNOSIS

These problem cases usually are seen by the surgeon as either (1) upper gastrointestinal bleeders, (2) cases of secondary hypersplenism, or (3) a combination of the two.

Basically, the diagnosis of portal hypertension is made by a high index of suspicions. The main lesion in the differential diagnosis is peptic ulceration; hence, an upper gastrointestinal series is indicated for its confirmation. The radiologic demonstration of esophageal varices is difficult, and the inability of the radiologist to demonstrate them should not be considered diagnostic of the presence or absence.

The presence of a palpable spleen may jar one's subconscious enough to be on the lookout for other diagnostic features of portal hypertension.

Endoscopic examination, such as esophagoscopic, or even gastroscopy, have been used. However, the risk of bleeding must be considered and prepared for by having blood available and a Patton tube handy.

The most reliable diagnostic test is the cessation of bleeding with esophageal tamponade.[18] Especially is this reliable if it oc-

Fig. 2.—Coarctation of inferior vena cava.

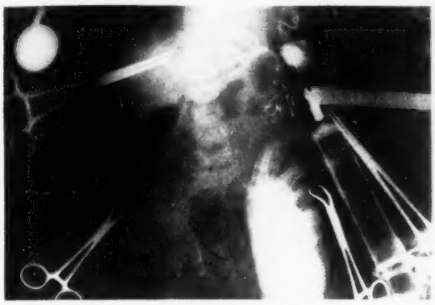

Fig. 3.—Splenoportagram of child 5 years old.

curs with clear return from the distal gastric lumen.

It has become progressively more evident that adequately to differentiate the hepatic group from the posthepatic group the portal system should be outlined. Percutaneous splenoportograms with 10 to 20 cc. of 70% iodopyracet (Diodrast) can be safely done shortly before or at surgery (Fig. 3). Such a procedure has been reported [5] in an 11-month-old infant, and in our last case (Table 2) such a procedure should have been carried out and was not done because of ignorance. Ignorance of what? There was a lack of knowledge that there are a number of patients in the posthepatic group who have strictures of the portal vein and who are, therefore, amenable to portocaval anastomosis. These patients are in the salvage group which has been neglected and can be attacked successfully at whatever age the strictures are discovered. It is important to note that of seven deaths reported by Shumacker [19] three of the four which came to postmortem examination had stenosis of the portal vein and only one had cavernomatous transformation. Two other cases of isolated stricture of the portal vein have been reported,[10] one proved by postmortem examination. However, to keep one completely humble, the presence of bleeding esophageal varices without hepatic or portal pathology has been reported by Mithoefer.[17]

SURGICAL THERAPY

Surgery for portal hypertension has progressed from splenectomy,[2] through shunt procedures † and transesophageal ligation ‡ to gastric resection [1] and interposed jejunal segments.[11]

† References 8, 9, and 22.

‡ Reference 3; G. Crile Jr. in discussion of Hollenberg and Briggs.[5]

TABLE 2.—*Nine Cases of Portal Hypertension in Infants and Children*

Case No.	Pathology	Age	Secondary Hypersplenism	Recurrent Bleeding	Bleeding Esophagus	Living	Shunt	X-Ray Varices	Portal Pressure, Cm. H_2O
1	Thrombosis, portal vein	14 mo.	0	+	Yes	Yes	Portacaval esophag. ligation	+	40.0+
2	Thrombosis, portal vein; cirrhosis	11 yr.	0	++++	Yes	Yes	Portacaval esophag. ligation presplenectomy	0	45.7
3	Cirrhosis	9 yr.	Yes	0	Yes	Yes	Splenorenal esophag. ligation	0	42.5
4	Thrombosis, portal vein	12 yr.	0	0	Yes	Yes	Splenorenal	0	45.0
5	Cirrhosis	13 yr.	Yes	0	0	Yes	Splenectomy splenorenal	0	30.0
6	Stricture	5 yr.	Yes	0	Yes	Yes	Splenectomy	+	33.0
7	Cavernomatous transformation, portal vein	12 yr.	No	++++	Yes	Yes	Splenectomy, 1947; total gastrectomy, 1952; Roux-Y	0	44.0
8	Cavernomatous transformation	3 yr.	Yes	++	Yes	Yes	Ligation of splenic art. & left gastric artery	0	13.2
9	Normal liver; stricture	17 yr.	Yes, in 1955	++++	Yes	Yes	Splenectomy, 1947; total gastrectomy, 1953; Roux-Y portacaval shunt, 1955	+	28.0

It is fair to state that in infants and children if an adequate splenorenal or portocaval shunt can be accomplished the low mortality and excellent clinical results are gratifying.§

Splenectomy as definitive treatment of portal hypertension in infants and children [19] or adults || can only be condemned. However, splenectomy alone for other than portal hypertension, if the portal vein has been found to be patent, is permissible, since the door is not then closed to a shunting operation if the occasion arises. Faced with the problem of a patient with portal hypertension

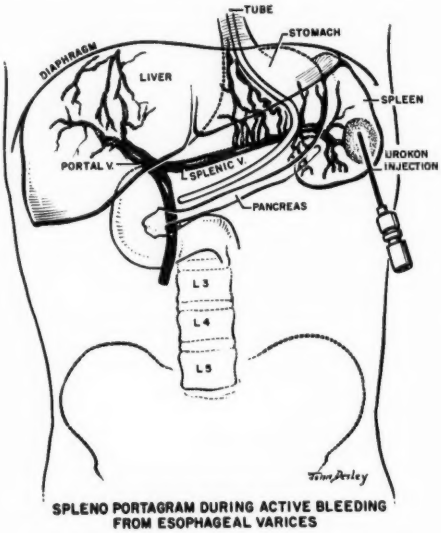

Fig. 4.—Splenoportagram of child 5 years old.

by direct measurement but no clinical evidence or history of bleeding, are you justified in doing a splenorenal shunt? We believe the answer is yes. Why? Because these patients usually have a normal liver which can easily stand the added strain of diversion of portal blood. To have such a patient develop bleeding and have obliteration of the portal vein is disastrous. Again it is clear that adequate diagnosis preoperatively by means of a splenoportogram would have solved the problem adequately.

The problem case is the patient with posthepatic portal-bed block, who, either like our first case (Table 2), is too young to have an adequate splenorenal anastomosis done and has a nonpatent portal for a portocaval vein or, like our second case (Table 2), has had a previous splenectomy without a splenorenal shunt and has a nonpatent portal vein. In these cases we used transesophageal ligation of the varices. The method is satisfactory to gain time and temporary control of bleeding—time to prepare and study the patient for a shunting procedure if possible. Its use as a definitive treatment we found wanting, since both of our cases subsequently bled again. This is in contrast to the belief of some who have treated a similar type of case.¶

The proponents # of acid peptic ulceration as a necessary factor in the etiology of bleeding esophageal varices have in this type of case (posthepatic) their best trial grounds. The most promising procedure, because of its relative low risk to the patient, is sphincter substitution by an interposed jejunal segment, as proposed by Merendino.[11] Esophagogastrectomy and devascularization of lower half of the esophagus combined with a vagotomy and gastroenterostomy were procedures found by Shumacker [19] to give poor results, owing to continual bleeding or high risk, or both.

Total gastrectomy or resection of the lower one-third esophagus and upper one-third of the stomach was found by Shumacker to carry a high mortality. In our two cases of total gastrectomy only one has had a satisfactory control of bleeding.

REPORT OF CASES

A general summary of our nine cases is given in Table 2, and a more detailed summary of the operative procedures, etiology, and mortalities in Tables 3 and 4.

CASE 1.—First admission, Dec. 29, 1953, of 5-month-old white boy with massive upper gastrointestinal bleeding. On Dec. 30, 1953, exploratory laparotomy was performed for bleeding duodenal ulcer; negative. Grossly normal liver.

§ References 19, 4, and 5.

|| References 21 and 2.

¶ Reference 3; G. Crile, Jr., in discussion of Hollenberg and Briggs.[5]

References 1-11.

TABLE 3.--*Operations in the Present Series*

Operations	Cases								
	1	2	3	4	5	6	7	8	9
Portacaval shunt	2	3	..	..	..	..	..	..	3
Splenorenal shunt	..	..	2	1	1	..	..	..	..
Sup. mes. vein to I. V. C. shunt	..	3	..	..	..	..	..	..	..
Splenectomy	..	1	2	1	1	1	1	..	1
Gastrectomy, total Roux-Y	..	..	..	..	..	..	2	..	2
Transesophageal ligation of varices	2	2	1	..	..	..	..	..	..
Ligation of left gastric art. and splenic art.	..	..	..	..	..	..	..	1	..
Exploratory laparotomy	1	..	..	..	..	..	..	..	..

0—done previous to this series

1—first operation.
2—second operation, etc.

TABLE 4.—*Summary of Nine Cases*

Pathology	
Cirrhosis	3
Thrombosis of portal vein or cavernomatous transformation	5
Stricture of portal vein	2
Hypersplenism	5
X-ray demonstration of varices	3
Range of portal pressure in cm. of H_2O	28 to 45
Recurrent bleeding after last definitive surgery	2
Age range	14 mo. to 17 yr.
Mortality, operative	0
Mortality, nonoperative	1

Second admission, Aug. 8, 1954, of a one-year-old child with anemia and hematemesis and melena. Palpable spleen and liver, ascites. Discharged Aug. 24, 1954.

Third admission, Sept. 14, 1954; well child. Sept. 20, 1954—a right thoracoabdominal incision was made. Cavernomatous transformation of portal vein found. Portacaval shunt done to good-sized varix also. Transesophageal ligation of varices done. Pathology on liver biopsy. Discharged Oct. 8, 1954.

Fourth admission, Dec. 29, 1954, of 17-month-old child. Coughed up some blood. High temperature. Hemoglobin 11.2 gm. Treated with antibiotics. Discharged on Jan. 3, 1955.

Comment.—Although this infant has returned twice to the hospital since his last procedure, at neither time was the bleeding massive. We believe that, even though his portacaval anastomosis was of poor quality, it must be working. It is anticipated that when the child is 3 to 4 years of age an adequate splenorenal shunt can be performed.

CASE 2.—First admission, Jan. 24, 1955, of 11-year-old boy. Well until 5½ years old; vomited blood. January, 1950, admitted to Mount Carmel Hospital and transfused. May, 1950, again admitted to Mount Carmel and transfused. November, 1952, splenectomy. April, 1954, again bled. Treated by transfusions 10 times. Dec. 29, 1954, again massive bleeding.

Patient's bleeding controlled with Patton tube, but could not be maintained; therefore, on Feb. 2, 1955, an exploratory laparotomy was performed. The liver was grossly cirrhotic—cavernomatous. Transformation of portal vein—portacaval shunt to varix about porta hepatis. A similar shunt to inferior vena cava to superior mesenteric vein done. This was also a poor shunt owing to size of vessels. Jan. 5, 1955—patient still bleeding and a transesophageal ligation done. Bleeding stopped for one week, only to begin to bleed off and on. Discharged after six months (June 22, 1955).

Second admission, Sept. 8, 1955. In coma and shock after bleeding for three days. On Sept. 30, 1955, patient died. Postmortem examination showed cirrhosis of liver with obliteration of portal vein, all shunts closed, and bleeding esophageal varices in upper and middle one-third of esophagus. Lower third obliterated by surgery.

Comment.—This is our only death, and at postmortem examination the patient had bleeding from esophageal varices in the upper and middle third of his esophagus. It is obvious that if at splenectomy an adequate splenorenal shunt could have been performed this boy would be alive today. An interposed jejunal loop should have been tried by us, but we missed our chance at his second operation and we never could get him into adequate preoperative condition.

CASE 3.—A 9-year-old Negro boy. On Feb. 6, 1955, he had a sudden onset of vomiting bright-red blood. Palpable spleen. Negative upper gastrointestinal series. Bleeding stopped temporarily with Patton tube but difficult to control.

On Feb. 14, 1955, ligation of esophageal varices and rapid exploratory laparotomy negative but for portal pressures of 43 cm. H_2O and enlarged spleen. Patient stabilized. Bleeding only once after this procedure. March 24, 1955, splenectomy and splenо-

renal shunt. April 3, 1955, discharged. July 30, 1955, no difficulty—seen in Outpatient Department.

Comment.—A straightforward case with an adequate splenorenal anastomosis and a gratifying result.

CASE 4.—February 14, 1955, admission of 12-year-old girl. Fainted at school and then vomited bright-red blood and then had two tarry stools later same day. Questionable palpable spleen. Hemoglobin 9 gm. Upper gastrointestinal series negative. Patient's bleeding controlled by Patton tube. Esophagoscopy negative on Feb. 23, 1955.

March 3, 1955, splenectomy and end-to-side splenorenal shunt. Spleen enlarged. Portal pressure, 468 cm. H_2O. Pathology—splenic fibrosis, negative liver.

Comment.—A case similar to Case 3 in a technical and result sense.

CASE 5.—A 13-year-old boy. June 1, 1955, followed one year in hematology clinic with diagnosis of Banti syndrome and secondary hypersplenism. He had a massive enlargement of spleen. Hemoglobin 5.5 gm.; RBC 3,500,000; WBC 1400; platelets 40,000; total proteins 6.8; thymol turbidity 11.7; cephalin flocculation 44; alkaline phosphatase 14.25; prothrombin time 17.4 sec.; control 12.6 sec. Patient transfused with fresh blood and on June 2, 1955, splenectomy of huge spleen done. Portal vein open. Portal pressure 30 cm. H_2O. Liver grossly cirrhotic. Pathology—cirrhosis of the liver, splenic fibrosis. June 12, 1955, patient discharged. Hemoglobin 12 gm.; WBC 12,000, platelets 664,650.

Comment.—As in Cases 3 and 4, we were well satisfied with the result.

CASE 6.—On May 23, 1955, a 5-year-old white boy was admitted with a two-week history of purpura; no other history, no gastrointestinal bleeding. Laboratory examination on admission: Hemoglobin 11.5 gm.; WBC 4750; platelets 110,000. Upper gastrointestinal series negative. Liver function test tuberculin precipitation 755. Albumin 4.75; thymol turbidity 120 units. Cholesterol 175 mg.; cephalin flocculation 2+; prothrombin time 15 sec.; control 12 sec. sulfobromophthalein (Bromsulphalein)—0% retention. Globulin 2.80; A/G 1.7/1.

June 27, 1955, splenectomy for a tri-portal spleen without one good splenic vein. Portal vein open and normal; liver grossly normal. Portal pressure 33 cm. H_2O. A Meckel diverticulum found and removed. Pathology: splenic fibrosis and normal liver.

July 5, 1955, discharged. Hemoglobin 10.6 gm.; WBC 11,200; platelets 353,000. To return for portacaval shunt.

Comment.—A splenoportagram should have been done when the patient was on the table to determine the etiology of this child's hypertension. The clinical course is excellent, but we believe he should have a portacaval anastomosis as soon as his parents will agree.

CASE 7.—This 12-year-old boy was admitted on June 30, 1952, to Jefferson Hillman Hospital after vomiting blood. He had had several previous attacks of hematemesis. Laboratory studies were essentially normal. Total gastrectomy performed July 9, 1952, after finding portal pressure of 44 cm. of H_2O. Liver appeared normal (the biopsy specimen taken was reported as normal liver), but the portal vein was not visualized. July 22, 1952, discharged.

Patient has not bled since this operation, and upper gastrointestinal series in 1953 showed no abnormal pathology of esophagus.

Comment.—A splenoportagram at operation would have been of value to determine the patency of the portal vein. This case has had an excellent result, but a shunt operation, if it could have been done, could have saved this boy's gastric pouch.

CASE 8.—A 3-year-old white girl. First admission: Oct. 13, 1953, at 19 months with complaint of hematemesis and melena with enlarged spleen and liver. Hemoglobin 7 gm.; platelets 73,680; prothrombin time 100%; cephalin flocculation 3+. Transfused and discharged with diagnosis of Banti's syndrome. Second admission: Feb. 17, 1955. Again hematemesis and melena. Spleen again palpable. Hemoglobin 7 gm.; platelets 253,000; prothrombin time 60%. Patient transfused, and on March 2, 1955, patient operated upon. Still bleeding. A splenoportagram done when the patient was on the table showed splenic and coronary veins but no portal vein. A gastrotomy revealed no bleeding inside the stomach. The spleen was large and the liver normal. Ligation of splenic and left gastric artery done because of the poor condition of the child on the table, and the abdomen was closed. Postoperatively she stopped bleeding and has done well.

Comment.—This child is to have a definitive shunting procedure when she has recovered sufficiently from her bleeding episode. She is about the minimal age for a splenorenal shunt, and with cavernomatous transformation of her portal vein she has only one chance to have surgical correction with the best results, i. e., splenorenal anastomosis.

CASE 9.—A 17-year-old girl. First admission, Jefferson Hillman Hospital, May 4, 1953, with complaint of gastrointestinal bleeding since the age of 2 years. During the next seven years repeated bleeding, usually hematemesis. Eight years previ-

ously, at age of nine years, splenectomy performed with diagnosis of Banti's disease. Admission laboratory studies: Hemoglobin 2.5 gm.; RBC 2,120,000 and WBC 19,960; sulfobromophthalein 28% dye retention in 35 minutes; clotting time 5½ minutes. She developed a lower nephron syndrome from blood transfusions but recovered and was discharged.

Second admission: June 1, 1953. Admitted for surgery, with RBC of 4,200,000; platelets 405,000; prothrombin time 100%. On June 5, 1953, she had a total gastrectomy with loop esophagojejunostomy and, 10 cm. distal, enteroenterostomy. Discharged on 16th postoperative day.

Third admission: Feb. 1, 1955. Complaint of 24 hours—had fainted and later passed a tarry stool. Laboratory studies: platelets 97,000; hemoglobin 4.5 gm.; WBC 7150; prothrombin time 60%. After transfusion patient was discharged to return for shunting operation in one month.

Fourth admission: March 7, 1955. Patient returned in good condition with hemoglobin of 13 gm., and Ba swallow showed varices down to the esophagojejunostomy. Sulfobromophthalein showed 48% of the dye retained; repeated and showed 6%. Prothrombin time 100%. Liver and spleen not palpable on physical examination. On March 14, 1955, patient operated on and a portagram done at the table through a tributary of the superior mesenteric vein showed a portal vein and many tributaries of portal vein coursing upward to lower end of the esophagus. A side-to-side portacaval anastomosis performed, with a thrill on release of clamps. Postoperative course uneventful and a Ba swallow eight days postoperatively could not demonstrate the previous large varices in the esophagus. Discharged on March 23, 1955. She has gained weight at home and has done well.

Comment.—This patient may represent a case of bleeding varices of the esophagus in which acid pepsin could have no part, and it demonstrates again that the best operation for portal hypertension, if it is possible, is a venous shunt.

SUMMARY AND CONCLUSION

In summary it can again be reemphasized that in portal hypertension of either hepatic or posthepatic origin shunting operations lead to almost universally good results. Splenectomy alone as a definitive treatment in this condition can only be condemned. This is particularly true when the pathology is posthepatic, since it removes the only possible shunting area.

The use of splenoportagraph is highly recommended in order to outline surgical therapy clearly and to find those cases of portal-vein stricture which may be adequately treated by portacaval shunt. This latter procedure, unlike a splenorenal shunt, can be performed at any age. A splenorenal vein anastomosis can, from a technical basis, best be performed above the age of four years.

If splenectomy is to be performed for other than portal hypertension and a hypertension is found at operation with obstruction of the portal vein, it is our belief that a splenorenal vein shunt should prophylactically be done.

From our clinical cases it would seem doubtful that in all cases of bleeding esophageal varices is acid pepsin digestion a necessary factor.

Liver coma from ammonium blood-level elevation has not been seen in children below the age of 12 years.

DISCUSSION

DR. JACOB K. BERMAN, Indianapolis: Drs. Jordan and Benson have asked me to discuss their stimulating paper, and I consider it a privilege to do so, albeit anything I may add to their comprehensive discussion would be entirely fortuitous.

In all discussions we should define our terms first, and here I wish to emphasize that we are speaking of extrahepatic portal obstruction and not that which is due to cirrhosis of the liver. Moreover, our nomenclature needs clarification. Dr. Jordan used the term "prehepatic obstruction," by which he meant an occlusion closest to the heart, that is, in the hepatic veins or vena cava. We have always used the direction of blood flow as the criterion for describing the site of vascular occlusion. In this sense the terms hepatopetal (centripetal) and hepatofugal (centrifugal) would connote pre- and posthepatic interference with the onward flow of blood toward the heart. It should be remembered, however, that the prehepatic occlusion may change the direction of blood flow, causing portal blood to drain hepatofugally into the systemic venous tributaries, as is exemplified in esophageal varices.

The etiology of bleeding varices is unknown, although the abnormality is generally associated with some form of portal obstruction. Yet esophageal varices do occur without portal or hepatic obstruction, and they often fail to occur when interference with portal flow is demonstrably present. Indeed, the mere presence of varices does not presage bleeding, and hemorrhage may occur without any tangible evidence of varix formation.

We cannot produce esophageal varices in dogs by slow occlusion of the portal vein, and if we tie the vein completely and suddenly the animals do

not survive. We have tried almost every conceivable method, including partial herniation of the liver into the thorax and complete intrathoracic translocation of the spleen, without success. However, varices may be produced in monkeys by combined methods of venous occlusion.

Why do esophageal varices form? Normally the esophageal plexus of veins drains into the azygos, the hemiazygos, the left and right phrenic, and the coronary veins. The left phrenic flows into the left suprarenal, and none of these veins have valves, except the azygos, where they are poorly developed. We are of the opinion that some of the venous blockade is in the outflow tract close to the varices as well as in the portal vein. Under these circumstances the esophageal plexus becomes greatly enlarged and tortuous, and, in addition to normal drainage, many adventitious channels develop because the higher pressure in the varices causes blood to seek an outlet in the systemic veins of lower hydrostatic pressure.

What makes the varices bleed? Is it the hydrostatic pressure in the vessel, negative pressure in the esophagus, mucosal peptic erosion, or a combination of these factors? It is unlikely, though not inconceivable, that peptic ulceration should occur in infants.

As the essayists have stated, many methods have been used to control bleeding. The esophagus has been injected, plicated, and partially and wholly excised. However, in the case that Dr. Jordan described, with varices up to the oral pharynx, I wonder if even total esophagectomy would help.

We have tried transesophageal ligations and the indirect methods, such as splenorenal shunts and paralysis of the diaphragm. I should like to tell you about a 17-year-old girl with extrahepatic portal venous obstruction. She had a splenectomy with a splenorenal shunt but continued to bleed periodically. She later developed pulmonary tuberculosis and was treated by pneumoperitoneum, thereby immobilizing both diaphragms. However, she had an esophageal hemorrhage while under treatment. After this experience it occurred to us that she was very much like a child with eventration, where the fundus of the stomach rises to a high level, producing angulation of the esophagus and obstruction of esophageal venous outflow. Our treatment at present must be regarded as empirical. Splenorenal shunts in infants function poorly because the vessels are too small and perhaps more collaterals are destroyed than any one shunt could overcome. Moreover, the spleen should be preserved unless diseased, because, after all, it is the largest single reservoir for portal blood and there are many spontaneous shunts around this reservoir.

Portacaval shunts in infants are often difficult to make because of cavernomatous transformations. I should like to know the long-term results of this procedure if anyone here has made such observations.

Dr. Arthur C. Pattison, Pasadena, Calif.: From a practical standpoint the problem of hemorrhage from esophageal varices in infants and children is no different from what it is in adults. There are a few variations.

First of all, an infant or child does not tolerate the massive hemorrhage as readily as does an adult. Second, the etiology of portal hypertension in infants and children usually is of the extrahepatic type—in other words, occlusion of the portal vein rather than the intrahepatic type of obstruction. Third, the size of the spleen in some of these infants and children as a result of portal hypertension is tremendous, and we have seen spleens that were so large that they extended into the pelvis.

Contrary to some of the statements a few years ago in respect to the differential diagnosis between the intrahepatic and extrahepatic types of portal hypertension, functional studies of the liver do not routinely give the answer. We have seen several instances now of cirrhosis that had absolutely normal liver profiles. Therefore, it is mandatory preoperatively to determine the site of the obstruction.

The splenoportagram, of course, is one excellent means of doing this. We approach the splenoportagram with some degree of apprehension, however, because it has been necessary on our service in one instance to do a splenectomy owing to a tear in the spleen as a result of the needle. Consequently, we now do them all with anesthesia and also do them immediately preexploration.

Another means of determining whether the portal hypertension is intra- or extrahepatic is that of the wedged-hepatic-vein pressure. Dr. D. C. Balfour Jr. and I two years ago at a meeting of this Society gave our initial report on this procedure. At the present time we have close to 200 patients upon whom wedged-hepatic-vein pressure readings have been taken for various situations involving the liver, and in only two instances of portal hypertension has there been an appreciable variation between the wedged-hepatic-vein pressure and the actual portal-vein pressure as measured at the time of operation.

Even in infants and children we are still content to use the Sengstaken tube to control the acute hemorrhage. Fortunately, we have not had to operate on any of the infants or children as emergencies. However, we have recently had our first failure in the use of a Sengstaken-Blakemore tube—failure in the sense that we were unable to control the hemorrhage in the patient, and he had to have his operation (a portacaval anastomosis)

done as an emergency. In three other instances, however, we have controlled the hemorrhage by a Sengstaken tube, only to find that upon release of the pressure bleeding would recur, and we have operated on these patients, doing shunts with the tubes in place.

I should like to substantiate one statement that Dr. Jordan made in regard to splenectomies. We believe splenectomy in portal hypertension should never be done unless one is doing a splenorenal anastomosis. In each of 13 instances now, in patients whom we have explored who previously had had splenectomies, we have been able to demonstrate a thrombosis involving the portal or splenic vein, oftentimes with enough recanalization that a shunt procedure could de done; however, in several instances such a complete occlusion made that procedure impossible.

Ammonium intoxication was mentioned. Our series of portacaval shunts is now 93 cases, and we have had two proved instances of ammonium intoxication, one which was readily controlled by diet; the second was that of a woman who could not tolerate as much as 20 gm. of protein a day without getting an elevated blood ammonia, and who subsequently died of hepatic failure.

I also was interested in Dr. Jordan's comment about inferior vena cava coarctation. One of the problems (not in children but in adults) that we have been faced with is the patient who does have portal hypertension but who also has an excessively high pressure in the inferior vena cava; so actually we are imbedding one high-pressure system into another system which also is much higher than it should be. In several of these instances we have been able to demonstrate a narrowing at the site of the inferior vena cava passing through the liver, and we have assumed that it possibly is due to contraction of the liver in producing the obstruction rather than an actual developmental coarctation.

Dr. Carl B. Davis Jr., Chicago: I apologize for rising again so soon but I should like to say how much I appreciated the paper of Dr. Jordan and Dr. Benson. We, too, have been interested in the problem of portal hypertension in children as well as in adults. The authors deserve much credit for their meticulous and painstaking care and the successful results they have reported in small children.

I should like to present very briefly the findings in a 7-year-old girl whom Dr. Fell operated upon one year ago, who, in 1951, had had a splenorenal shunt performed at the age of 4 years at another hospital. There was recurrence of hematemesis all too soon after the splenorenal shunt. The probabilities were that thrombosis and closure of the anastomosis occurred, although no splenogram was done to determine this.

We, like Dr. Pattison and Dr. Berman, believe that splenoportagrams are not without risk.

[Slide] In this first x-ray you will note on the left the barium examination of the esophagus at age 4, which demonstrates the extent of involvement of the esophagus by varices. The center film shows the condition of the esophagus three to four months after the splenorenal anastomosis. The x-ray on the right shows the esophagram six months prior to the operation performed at our hospital. Because of continued episodes of bleeding, with hemoglobin levels down to 4 gm. and a red blood count down as low as 1,000,000 cells, additional surgery was recommended.

[Slide] After adequate blood replacement and the usual preoperative preparation, through a right combined thoracoabdominal incision a portacaval anastomosis was accomplished. Note the collateral or accessory vein, which in the presence of an atretic portal vein was joined end-to-side to the inferior vena cava. The liver was normal.

[Slide] This shows the esophagram nine months after the portacaval anastomosis. No varices are seemingly apparent. The most interesting and important point regarding this case is that this patient, now 8 years of age, has not bled once since this last operation.

I should like to direct one question to Dr. Jordan. We have under our care at the present time a 4-year-old boy who has had hematemesis presumably on the basis of an extrahepatic block. The spleen becomes markedly enlarged, and bouts of hematemesis occur only at times when this patient experiences an upper respiratory infection and develops fever.

Are we doing this boy an injustice by postponing surgery until the time when his vessels will become larger for performing an adequate anastomosis, or should we be more aggressive and treat him surgically in the very near future? If the latter is true, what type of procedure would you recommend?

Dr. John H. Grindlay, Rochester, Minn.: When a complete shunt of blood from the portal vein into the inferior vena cava is made in dogs, there is progressive loss of weight and vigor and finally coma and death. Such has been the experience of all who have made complete Eck fistulas in dogs, and this is the reason why many of us have had theoretical objections to the operation of portacaval shunt for "portal hypertension" in man. We feared that the operation might have an adverse effect on the liver, might even precipitate hepatic coma.

Some recent studies in our laboratory, in which one of our fellows, Dr. Frank Lichtenheld, now of Wiesbaden, Germany, worked with my colleagues, Drs. J. L. Bollman, G. A. Hallenbeck, and R. G. Bickford, and me, prompt me to add a remark to the paper of Drs. Jordan and Benson. My remark

is that there is a great difference in the effect of a complete portacaval shunt (Eck fistula) on a previously normal dog and on a dog with an experimentally induced, chronic occlusion of the portal vein at the hilus of the liver.

Our studies have shown that the latter animal does not develop the "Eck fistula syndrome." The animal remains in the same state as it was before the shunt was made; coma does not develop, and the animal does not suffer from further loss of weight and vigor. It has been of interest to us, also, that in these dogs the degree of engorgement of the portal system of veins and of size and number of collateral veins does not seem to be affected by an end-to-side portacaval anastomosis.

DR. PRESCOTT JORDAN, Detroit: I wish to thank all of the discussants.

There is one other problem that no one has touched upon and that is that if a child has secondary hypersplenism and you do a portacaval shunt on him, will the secondary hypersplenism due to the engorgement of the spleen go into remission? I have been unable adequately to test this clinically, but I notice that other people have thought of it; however, the question has not been answered.

To answer Dr. Davis's question, I am sure I can be of no help. I am afraid that if I were presented with it myself I would do a splenoportagram, thinking that that child possibly at 4 years of age may have a stricture of the portal vein instead of a cavernomatous transformation; if he did, then a portacaval shunt should be done at any age, and if he didn't then I believe a more conservative method of teasing him along until you could get the only remaining shunt area left in his body (a splenorenal shunt that would be adequate to carry him on and prolong his life) would be the treatment of choice.

REFERENCES

1. Baronofsky, I. D.: Portal Hypertension, with Special Reference to Acid-Peptic Factor in Causation of Hemorrhage and Extensive Gastric Resection in Its Treatment, Surgery **25**:135, 1949.

2. Cole, W. H.; Walter, L., and Limarzi, L. R.: Indications and Results of Splenectomy, Ann. Surg. **129**:702, 1949.

3. Crile, G., Jr.: Transesophageal Ligation of Bleeding Esophageal Varices: Preliminary Report of 7 Cases, Arch. Surg. **61**:654, 1950.

4. Gross, R. E.: The Surgery of Infancy and Childhood, Philadelphia, W. B. Saunders Company, 1953.

5. Hollenberg, H. G., and Briggs, B. P.: Portal Caval Anastomoses in Infants, Ann. Surg. **141**: 648, 1955.

6. Keith, A.: Human Embryology and Morphology, Ed. 4, Glasgow, Glasgow University Press, 1923.

7. Klemperer, P.: Cavernomatous Transformation of Portal Vein: Its Relation to Banti's Disease, Arch. Path. **6**:353, 1928.

8. Linton, R. R.: The Surgical Treatment of Bleeding Esophageal Varices by Portal Systemic Venous Shunts with Report of 34 Cases, Ann. Int. Med. **31**:794, 1949.

9. Linton, R. R.: The Selection of Patients for Portacaval Shunts, Ann. Surg. **134**:433, 1951.

10. Mahoney, E. B., and Hogg, L., Jr.: Congenital Stricture of the Portal Vein, Arch. Surg. **61**:713, 1950.

11. Merendino, K. A., and Dillard, D. H.: The Concept of Sphincter Substitution by an Interposed Jejunal Segment for Anatomic and Physiologic Abnormalities at the Esophagogastric Junction, With Special Reference to Reflux Esophagitis, Cardiospasm and Esophageal Varices, Ann. Surg. **142**:486, 1955.

12. Meyer, A.: On a Case of Complete Fibrous Obstruction (Congenital?) of Both Superior and Inferior Venae Cavae in a Young Man of 18 Years —First Recorded Case, M. Rec. **63**:806, 1903.

13. Bennett, I. L., Jr.: A Unique Case of Obstruction of the Inferior Vena Cava, Bull. Johns Hopkins Hosp. **87**:290, 1950.

14. Huseby, R. A., and Boyden, E. A.: Absence of the Hepatic Portion of the Inferior Vena Cava with Bilateral Retention of the Supracardinal System, Anat. Rec. **81**:537, 1941.

15. Keen, J. A.: The Collateral Venous Circulation in a Case of Thrombosis of the Inferior Vena Cava, and Its Embryological Interpretation, Brit. J. Surg. **29**:105, 1941.

16. Osler, W.: Case of Obliteration of Vena Cava Inferior, with Great Stenosis of Orifices of Hepatic Veins, J. Anat. & Physiol. **13**:291, 1878-1879.

17. Mithoefer, J.: Bleeding Esophageal Varices Without Other Signs of Hypertension in a Child, J. A. M. A. **153**:1097, 1953.

18. Patton, T. B., and Johnston, C. G.: A Method for Control of Bleeding from Esophageal Varices, Arch. Surg. **59**:502, 1949.

19. Shumacker, H. B., Jr., and King, H.: Splenic Studies: Portal Hypertension in Children Associated with Gastroesophageal Hemorrhage, A. M. A. Arch. Surg. **65**:499, 1952.

20. Rotnoff, O. D.; Cauley, C. L., and Berthrang, M.: The Difference Between Extra- and Intra-Hepatic Obstruction of the Portal Circulation, Bull. Johns Hopkins Hosp. **87**:305, 1950.

21. Rousselot, M.: The Late Phase of Congestive Splenomegaly (Banti's Syndrome) with Hematemesis but Without Cirrhosis of the Liver, Surgery **8**:34, 1940.

22. Whipple, A. O.: Problem of Portal Hypertension in Relation to Hepatosplenopathies, Ann. Surg. **122**:449, 1945.